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VOL. XLV. No. 5

ARCHIVES OF OPHTHALMOLOGY.

STUDIES OF OPTIC-NERVE ATROPHY IN ASSOCIATION WITH CHIASMAL LESIONS.

BY CLIFFORD B. WALKER, M.D., AND HARVEY CUSHING, M.D.

(From the Surgical Clinic, the Peter Bent Brigham Hospital, Boston.)

(With 47 cuts in the text and on Text-Plates XII–XXIII.).

I. INTRODUCTORY.

IN a recent communication (8) a series of cases were recorded to illustrate what we have come to designate as stages in the development of hemianopsia when produced by the pressure of hypophysial or parahypophysial tumefactions against the chiasm. It was pointed out (1) that in 81 examples of field deformation from these lesions they proved to be bitemporal in 26, homonymous in 12, and unclassified in 8, while in 35 there was blindness in one or both eyes, so that it was uncertain in which group, bitemporal or homonymous, they belonged; (2) that in cases of bitemporal hemianopsia it is unusual for the field defect to be bilaterally symmetrical; (3) that in the majority of the cases the first perimetric indication of the process when the usual 5*mm* test objects are used is shown by a slant in the boundary of an upper temporal field and a corresponding quadrantal defect in the color peripheries. The advance of this temporal-field defect downward with ultimate involvement of the macular area and encroachment on the nasal field was divided, for convenience of clinical

designation, into eight stages.[1] It was shown, moreover, that the recession of the defect after operative measures with relief of pressure took place more or less rapidly and completely in a reverse sequence.

In another place, one of us (Walker, 30) described some of the more irregular cases in which the defect proceeds from below instead of from above, or from above and below at about the same rate, and recently we have seen a patient in whom a temporal defect was about equally advanced from above in one eye and from below in the other. Furthermore, in determining the presence of cæco-central scotomata and temporal islands, as well as in plotting the early changes in the peripheral fields, attention was called to the use of a series of test objects increasing in size from that representing the smallest available visual angle (1.5 min.) to the largest satisfactory size (8 degrees). Whether the variation of the field defect represented a variation of visual fiber bundles in the chiasmal region or variations in the asymmetry of the tumefaction could not be definitely determined, though the latter cause was favored.

At the conclusion of our last conjoint paper (8) the question of primary and secondary optic atrophy was briefly discussed and surmises were made as to the position of the fascicular fibers in the optic path and the order in which atrophy occurs in them. It was stated that it was often impossible to tell from the appearance of the optic disk whether or not there had been an irrecoverable destruction of the nerve; indeed, vision had been seen to return in blind eyes even when pupillary reaction had also been lost. Thus, as has been previously emphasized by one of us (Cushing, 7), the early perimetric defects are to be regarded rather as an evidence of physiologi-

[1] We were not the first to call attention to this characteristic progression of visual defects in cases of pituitary lesion. Dr. Arnold Josephson was possibly not only the first to demonstrate the enlargement of the sella turcica by Röntgenographic methods in a case of acromegaly, but he also observed in some cases the form of advancing perimetric defect which begins in the upper temporal quadrants. Unfortunately for us, there has been no translation into English of Josephson's admirable monograph (*Studier öfver Akromegali och Hypofystumörer*, Stockholm, 1903), and we are glad to credit him at his request with the priority which he deserves. J. Herbert Fisher, as we pointed out, had also noted the condition.

cal block than of an anatomical destruction of the nerves, in view of the great degree of recoverability in the transmission of light impulses on the release of the nerves from pressure. This was further supported by the fact that in several cases the histological examination of nerves showed but a scattered degeneration, even though the ophthalmoscope had previously demonstrated the characteristic pallor of advanced atrophy, and the perimeter an advanced field defect.

Since the classical study of Henschen (18) on the optic pathways did not include examples of chiasmal lesions such as we were given the opportunity to study, we wished to see whether or not the tract degenerations in our cases conformed with those which he had observed. For the interruption in the pathway in Henschen's cases was produced chiefly by cerebral tumors, capsular hemorrhages, or other vascular lesions of long duration, varying from one to fifteen years—in other words, by lesions central to the chiasm. In these long-standing cases, the secondary degeneration in the optic nerves had become well defined and although the degenerated areas still showed a scattering of undegenerated fibers, they nevertheless formed a striking contrast with the rest of the nerve. In posterior lesions of this type, one tract is interrupted entirely, while the other is functionating normally. Hence they tend to give clean-cut field defects which are practically the same for small and large disks.

The field defects from chiasmal lesions, on the other hand, when plotted with graded disks, indicate that the interruption of the nerve fibers is much more diffuse in distribution, for larger disks give a proportionately wider field. Thus, from the character of the fields, from the anatomical position of the lesion, and from the character of the onset of the hemianopsia, one might expect to find a more diffuse process in anterior than posterior lesions. Indeed, it seems remarkable that a pituitary struma can eventually produce a stage of vertical hemianopsia in each eye with both crossed and uncrossed fasciculi remaining under the same degree of pressure from the start.

It was our original view that pressure against the chiasm with resulting distension was the chief element in producing the field defects, but an instrument which has been devised

by one of us (Walker), in which wires could be woven in a way similar to the chiasmal arrangement, has led us to believe that tension as well as pressure is an important factor, and we may add that Fisher (15) also reached a similar conclusion.

Material and Methods. In a series of some two hundred operations upon patients with hypophysial tumors, there have been at this writing eight fatalities, most of them occurring in the earlier series at a time when unfortunately we employed less exact methods of perimetry than at present. However, fields were plotted in all of these cases, except those already blind, on one or more occasions shortly before the operation, so that an opportunity was afforded to compare the field defects, which indicated the degree of functional disturbance, with the degree of actual fiber degeneration in the nerves existing at the time. In none of these cases was there any ophthalmoscopic evidence of the secondary changes due to a choked disk, though in some of them a low grade of recent œdema was superimposed on the picture of so-called primary atrophy.

We had hoped that by subjecting this material to accurate histological studies of the degenerated fiber bundles we might be able to supplement Henschen's maps of the course which the fasciculi from the retinal quadrants pursue through nerve, chiasm, and tract. We soon found, however, that in many of our cases the atrophy in the optic nerve was either too slight to be well traced or, in more advanced cases, was too diffuse. As a matter of fact, the chiasm and tracts were often so flattened and distorted by the tumor that a sectional survey of the normal anatomical situation of particular bundles which might have undergone excessive degeneration would have been impossible.

We found, too, that it is very difficult, without loss of orientation, to carry sections of the nerves having poorly demarked tracts through the preliminary steps necessary for microscopic study. This is particularly true of the orbital portion of the two optic nerves, which in the process of removal are necessarily detached from the intracranial portion of the pathway including chiasm and tracts. Knowing which was right and which was left nerve, however, it was possible by the aid of the macula to identify the upper and lower, inner and outer aspect of each nerve, and the block that was to be removed for

sectioning was previously nicked with a sharp knife, doubly on the lower and singly on the outer aspect of Schwalbe's sheath. These marks, when traced in India ink, served sufficiently well for the subsequent orientation of the mounted sections. The blocks, moreover, were numerically marked according to the position in the nerve from which they had been cut, as indicated in the figure (Fig. 1), which will serve

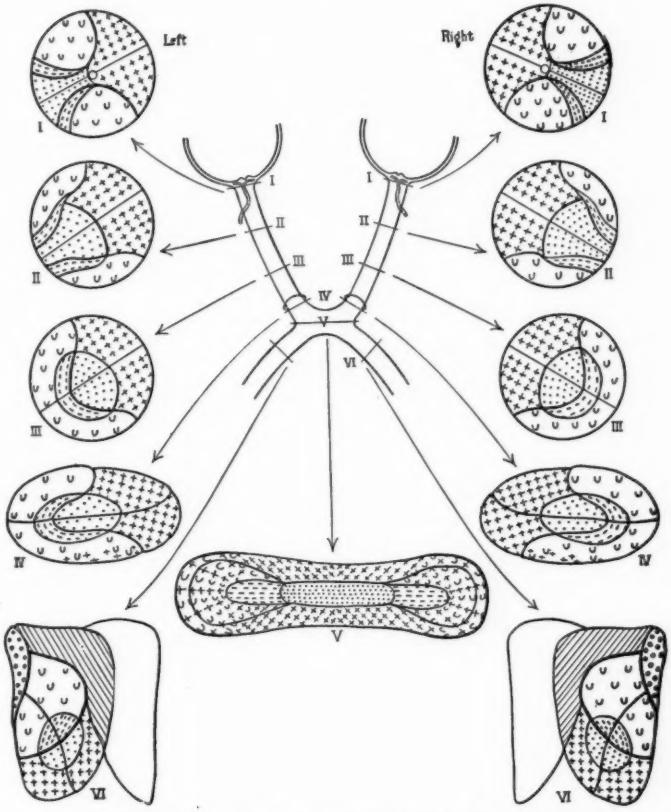

FIG. 1.—Diagram of nerves, chiasm, and tracts to indicate where sections have been taken; also Henschen's fascicular distribution at these levels for both right and left nerves seen from behind.

roughly to identify the anatomical position from which the sections to be reproduced were taken, and which for purposes of orientation will also indicate on cross-section the fascicular distribution of Henschen for each particular region in each nerve.

II. CASE REPORTS

In our eight cases there are examples which run all the way from mild degeneration to states (*cf.* Case 8) in which there are comparatively few histologically normal fibers remaining.

Although our cases differed from those of Henschen, in that the lesion was anterior and comparatively recent rather than posterior and of long standing, still we anticipated no difficulty in identifying in the nerve sections the degenerated fasciculi, involvement of which was indicated by the character of the perimetric defects. But we found that this relation of field defect to fascicular atrophy was far less definite than we had expected and in many instances was difficult to determine.

The following case, which may be cited in illustration, shows that after a complete blindness of six and one half months' duration in one eye, following a bitemporal hemianopsia which had persisted for nine or ten months, the amount of atrophy found in the optic nerve was hardly noticeable.

CASE 1.—(P. B. B. H. Surg. No. 789.) *Interpeduncular cystic tumor with bitemporal hemianopsia advancing to blindness. Temporary restoration of vision after operation. Death. Examination of nerves.*

January 11, 1914. Admission of Mrs. Anna S., aged 41, referred by Dr. Sherman Voorhees of Elmira, N. Y.

She has had good health until *August, 1913*, when after a severe headache with vomiting she found that her vision was blurred and that she saw double. Following this her menstruation ceased and she thought herself pregnant. On *November 15, 1913*, Dr. Voorhees found a bitemporal hemianopsia. Since *December 25th* the left eye has been blind. She has gained twenty pounds in weight but there are no other clinical endosecretory manifestations. The X-ray shows a normal sella. There have been definite psychoses suggesting frontal lobe involvement.

Examination of Eyes. No exophthalmos, ptosis, or squint. Movements normal but with slight nystagmus. *Pupils* slightly unequal, left larger than right; react together to light and accommodation, but the left reacts very sluggishly, if at all, to light and consensually. Direct pupillary reaction on the left nil, indirect normal. *Fundi*: O. U., disk margins clear but not narrowed; optic cup present, moderate depth; lamina cribrosa sharp. Disk pale but not glistening. Pallor more marked on left than on right. Slight nasal hyperæmia. Peripheral fundus normal. *Fields* (Fig. 2) show complete blindness on the left and tem-

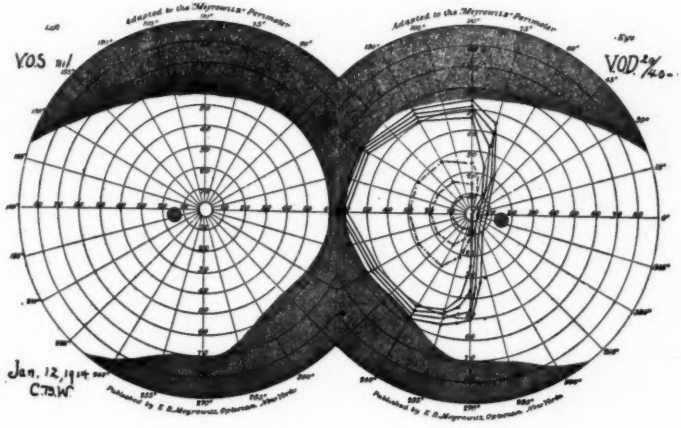

FIG. 2.—Case I. Fields of Jan. 12, 1914, on first admission. O.S. Stage VIII; O.D. Stage IV. (Inner form field = 0.5*cm* disk.)

poral hemianopsia in Stage IV on the right. V. O. S. nil; V. O. D. $\frac{20}{40}$. Hemiopic pupillary reaction present on right.

Clinical Diagnosis: Suprasellar (interpeduncular) tumor or cyst.

January 17, 1914. Operation I. Right subtemporal decompression. Very slight tension revealed.

January 28th. No improvement in condition. Decompression area remains collapsed. Discharged.

March 15th. Readmission owing to recent progressive failure in vision, associated with protrusion of decompression area. Pupils equal and dilated. Direct pupillary reaction present on the right, absent on left. *Disks* show increased

pallor. *Fields* (Fig. 3) show an advance on right to Stage VII. V. O. S. nil; V. O. D. $\frac{2}{200}$. Marked increase in mental symptoms with drowsiness.

March 20th. Operation II. Orbito-frontal approach with osteoplastic flap. Evacuation of large interpeduncular cyst and removal of fragment of wall of cyst, which showed characteristic pharyngeal squamous epithelium.

On recovery from the anæsthetic it was found that vision had returned in the left and on the nasal fields of the right, and though after forty-eight hours the sight had faded from the

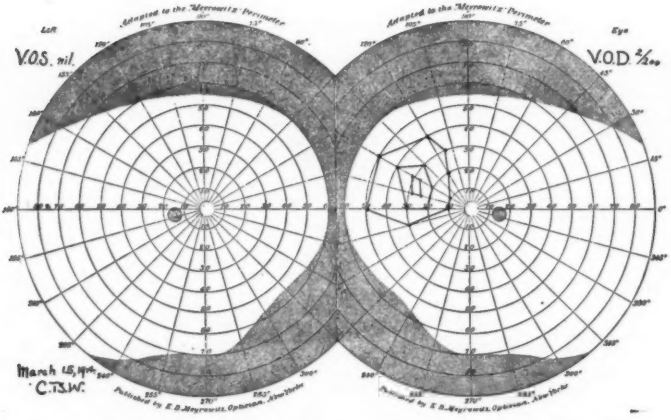

FIG. 3.—Case I. Fields of March 15, 1914, five days before second operation, showing advance in right eye to Stage VII. (Inner form field = 1.0*cm* disk.)

left eye it persisted on the right until the patient was up and the fields could be plotted.

April 1st. Fields show typical right hemianopsia (as in Fig. 4). V. O. S. nil; V. O. D. $\frac{1}{10}$. *Fundi* show no change.

April 13th. The restored vision on the right has again been rapidly failing, and three days ago light perception entirely disappeared. She is *at present totally blind* though the pupils react sluggishly to a bright light.

April 17th. Operation III. Second orbito-frontal operation. The bone flap was re-elevated and the chiasmal region was again exposed, with a second evacuation of the cyst, which had completely refilled. An attempt was made to permanently drain the cyst by the placement of linen threads.

On recovery from the anæsthetic it was found for a second time that vision had returned in the right eye.

April 23d. Fields as in Fig. 4. V. O. S. nil; V. O. D. $\frac{20}{50}$.

April 25th. Field narrowing. V. O. S. $\frac{20}{100}$.

April 26th. Again total blindness.

She remained completely blind for the following ten days. Realizing that the procedures by the subfrontal route were ineffectual, an attempt was made to enter and drain the cyst by a callosal puncture.

May 12th. Operation IV. Callosal puncture. After primary puncture of the lateral ventricle with the disclosure

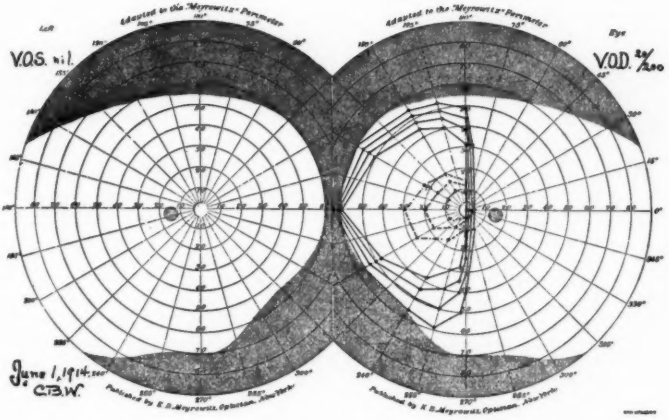

FIG. 4—Case I. Fields plotted June 1st, after the third post-operative return of right nasal vision. (Inner form field = 0.5*cm* disk.)

of a suspected internal hydrocephalus, the usual callosal puncture alongside the falx was made, the trocar being inserted well into the region of the third ventricle. There was no prompt return of vision as on the former occasions, but by *May 18th* she was able to recognize attendants, to count fingers, and to tell colors. Fields show slight nasal vision on the right. V. O. D. $\frac{5}{100}$. From this time on there was continued improvement, and by—

June 1st. The fields (Fig. 4) showed a condition about comparable to that on her first admission (*cf.* Fig. 2), though the vision was lowered. V. O. S. nil; V. O. D. $\frac{20}{200}$.

Seven days later, with evidences of great increase in intracranial tension, she became stuporous and a ventricular

puncture was again performed, which did not serve to relieve the symptoms. Death.

Autopsy. Brain fixed *in situ.* A large suprasellar cyst occupying the interpeduncular space was disclosed (Fig. 5). It measured about 5*cm* in diameter and was lined with typical verrucous nodules of squamous epithelium. Of the chiasmal structures the tracts had chiefly suffered from the pressure, they being thinned to mere ribbons of tissue.

On *histological examination* the optic nerves show surprisingly little degeneration of the fibers, even on the left side. It is notable that what little atrophy is present seems much more marked in the vessel-bearing portion of the nerve than in the vessel-free portion. The vessel-free sections (Figs. 6 and 7) have a normal appearance and show under the high power only a very slight scattering of degenerated fibers, possibly somewhat more marked in the crossed and macular tracts than in the uncrossed. In the vessel-carrying sections (Figs. 8 and 9) the same relations hold, except that the amount of degeneration seems a little greater. It is also of interest that the retinal ganglion cells in these eyes showed no degeneration, according to the report of Dr. F. H. Verhoeff, who had the kindness to examine these sections with one of his delicate staining processes.

Comment. This case furnishes a good clinical example of how the nerves may be affected by a physiological block to the transmission of impulses long before an anatomical destruction of the fibers occurs. On the basis of perimetric estimates each eye had progressed through the typical stages of a temporal hemianopsia to blindness, and yet, after the successful evacuation of the cyst, vision promptly returned in each eye with recession to Stage IV—one of the eyes (left) having been totally blind for six and one-half months. For the better of the two eyes, this procedure was repeated with return of vision again, and still a third time, though on the last occasion, after ten days of blindness, the return of vision was less abrupt, possibly as the immediate pressure release was less complete after the callosal puncture than when the cyst had been widely opened after a direct exposure.

In contrast to the insignificant evidence of atrophy in the nerves, the marked gross deformation of the tracts, which were

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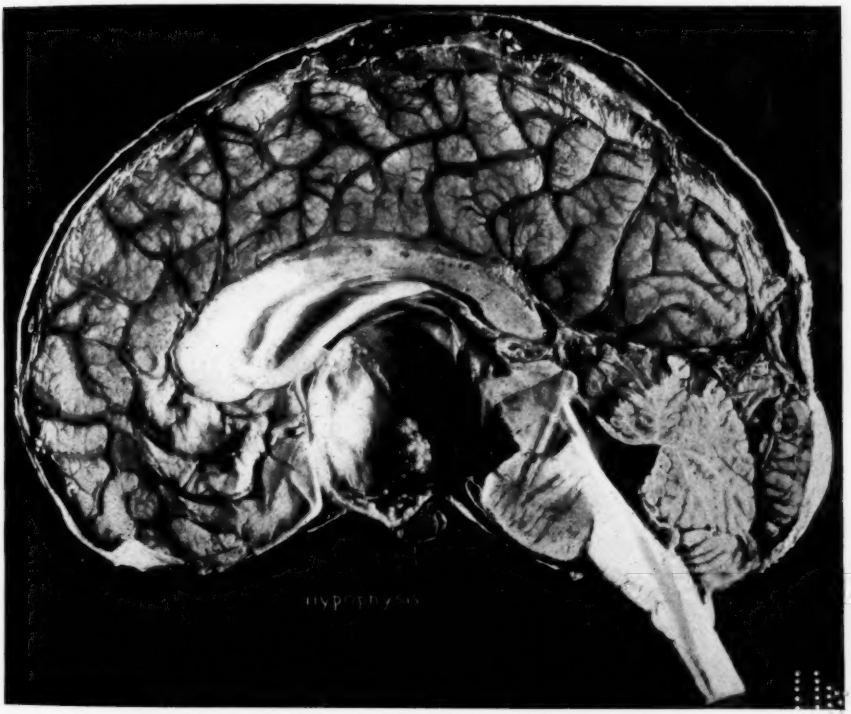

FIG. 5.—Case I. Sagittal section of brain showing large suprasellar cyst lined with verrucous nodules

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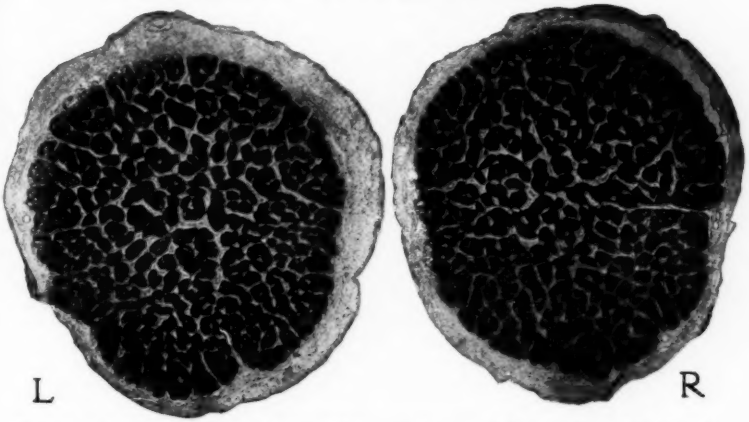
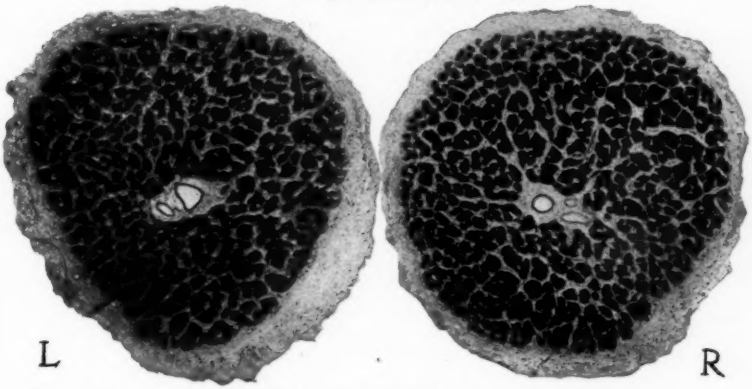
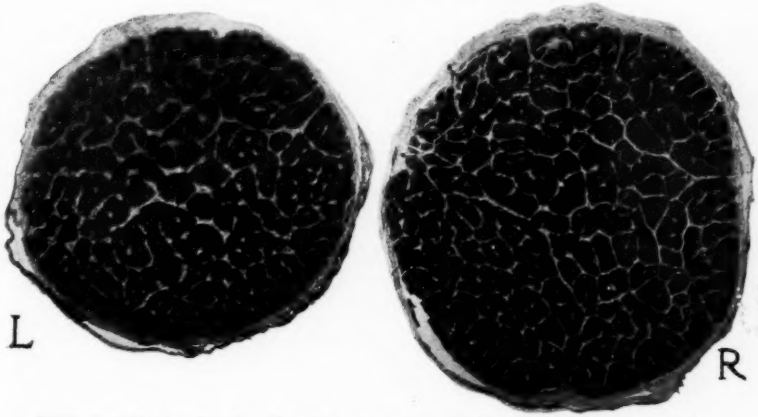


FIGS. 6 and 7.—Case I. Sections of the posterior vessel-free portion of both optic nerves. The very slight atrophy of the crossed and macular bundles is apparent only under the microscope.



FIGS. 8 and 9.—Case I. Showing sections of right and left nerves in the vessel-bearing segment. Most apparent degeneration in the crossed and macular bundles, particularly of the right nerve.



FIGS. 12 and 13.—Section of optic nerves in vessel-free portion showing slight diffuse degeneration on the left.

not sectioned, must be emphasized especially in view of the findings in the next case in which, like the above, the evidences of degeneration in the nerves were insignificant, even though there was total blindness in one eye.

CASE 2.—(J. H. H. Surg. No. 27100.) *Huge hypophysial struma distorting the brain. Primary hypopituitarism, with neighborhood, general pressure and endosecretory symptoms all pronounced. Sella destroyed; hemianopsia; choked disk on primary atrophy; adiposity, feminism, genital dystrophy. Subtemporal decompression. Death. "Status lymphaticus."*

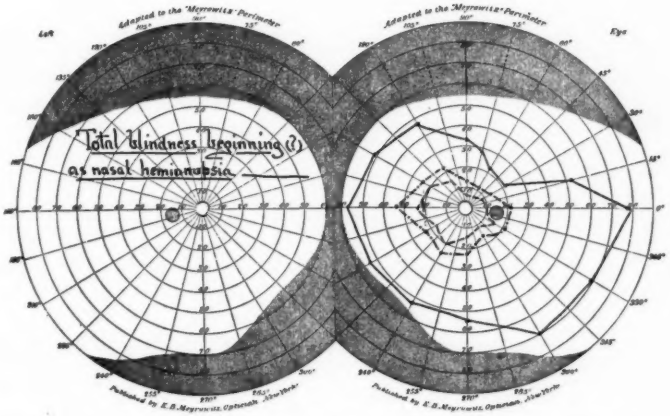


FIG. 10.—Case II. Fields (0.5*cm* disk) of Dec. 22, 1910, on admission.

Dec. 21, 1910. Admission of H. G. W., aged 22, referred by Dr. M. Allen Starr of New York. The clinical history of this case has already been recorded in full[1] and for our present purposes it may merely be said that on the patient's admission there was total blindness, with atrophic pallor of the left disk, in which the first perimetric change, noticed seven months before, had been a nasal hemianopsia. The right eye, which showed a low grade of choked disk, was in an early stage of a temporal hemianopsia (Fig. 10).

The patient died after a subtemporal decompression undertaken for the relief of his marked general pressure symptoms. The autopsy disclosed a large hypophysial

[1] Cushing, H., *The Pituitary Body and Its Disorders*, Case 5, p. 48.

adenoma (Fig. 11) which had extended far into the third ventricle and into each temporal lobe.

Discussion. The nerve on the left (Fig. 12), the blind side, is smaller than the other and shows histologically some diffuse degeneration; the nerve on the right (Fig. 13) shows none.

The duration of blindness and the condition of the vessel-free portion of the optic nerves were very similar in this case and in the preceding one. In Case 1, however, since the chiasm and tracts were reduced to mere ribbons the histological examination was fruitless, but here, though they were undisturbed by the tumor and appeared normal, nevertheless, as would have been presumed, they showed more marked degeneration than did the nerves. For it is natural that with chiasmal pressure, atrophy first appears in the tract as a peripheral degeneration with respect to the retinal ganglion-supporting cell and the point of pressure.

A section midway in the anterior third of the tracts (Figs. 14 and 15) shows this phenomenon very well. The sections are cut somewhat slanting and viewed from the front. In the right tract (Fig. 15) the crossed fibers, and in the left tract (Fig. 14) the uncrossed fibers show definite degeneration. Meynert's commissure is well made out as an additional landmark. The line of demarkation between the crossed and uncrossed bundles is hazy, owing to the characteristics pointed out by Henschen.

This patient was blind in the left eye with the right temporal field beginning to show involvement, and we hoped to determine whether the process originated as a homonymous or a bitemporal one. If the latter, the crossed fasciculus in the right tract (= temporal field) should show a more advanced degeneration than the uncrossed fasciculus from the left tract to the same eye (= nasal field), but this from the sections cannot clearly be determined. This would indicate, perhaps, that no great interval of time had elapsed, inasmuch as the slight temporal defect in the right eye had already produced a corresponding area of atrophy in the ventral crossed bundle of the left tract.

In these two cases, then, the loss of myelin about the optic-

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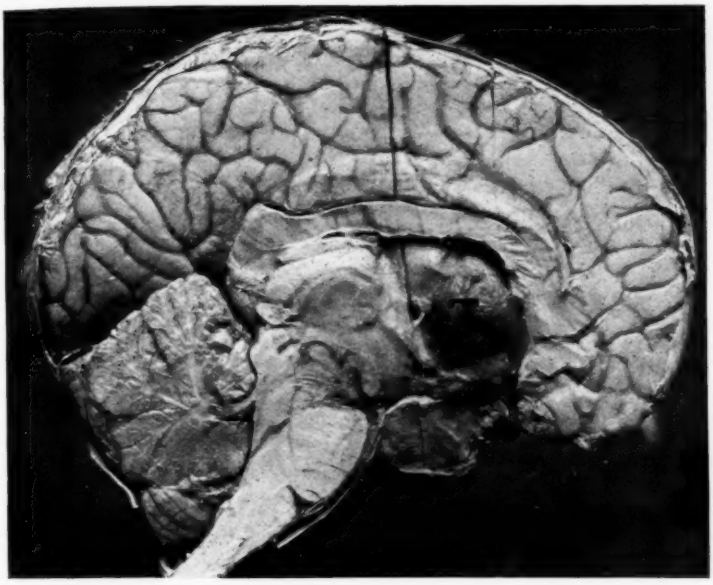
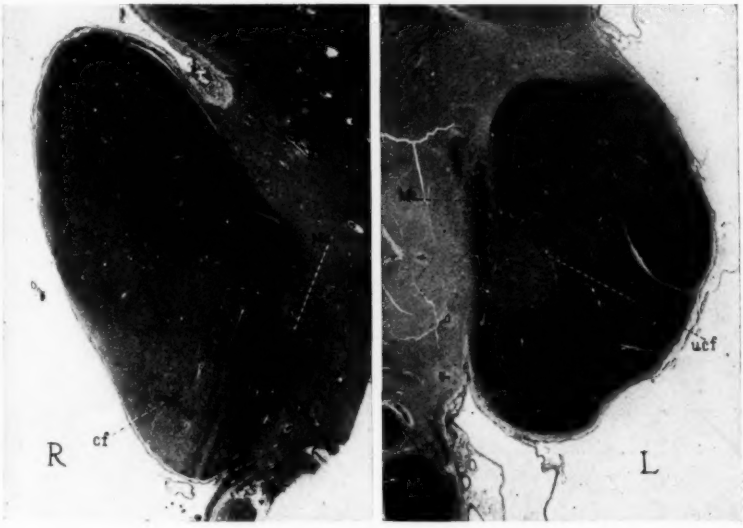


FIG. 11.—Case II. Showing pituitary enlargement with marked intracranial extension (T).



FIGS. 14 and 15.—Sections seen from the front (Fig. 1, Sec. VI. reversed), taken midway in tract and cut somewhat slanting. Showing degeneration in right crossed (cf) and left uncrossed fasciculus (ucf). Meynert's commissure (Mc).

nerve fibers seems to have taken place first in the tract, and there maintained its lead over myelin loss in the nerve throughout. In this respect the optic nerve does not differ from other nerves, which commonly atrophy first in their portions distal to the point of pressure or functional interruption.

The next figures represent the condition of the nerves in a case of complete right homonymous hemianopsia with a vertical meridian, and considerable peripheral contraction of the remaining field.

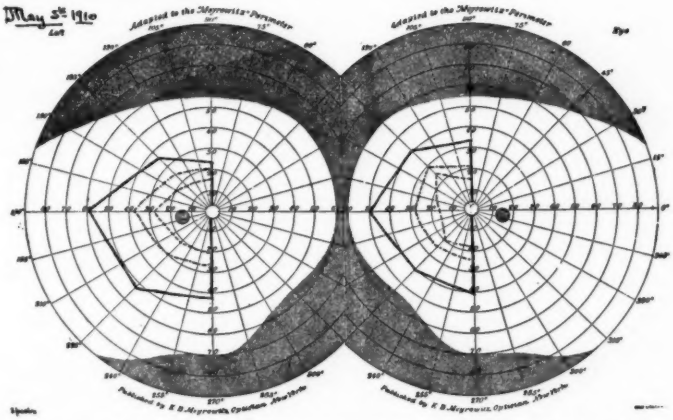


FIG. 16.—Case III. Fields taken a few days before death, showing right homonymous hemianopsia (0.5*cm* disk).

CASE 3.—(J. H. H. Surg., No. 25971.) *Acromegaly with an actively growing chromophobe struma producing pronounced neighborhood as well as general pressure symptoms. Operation. Death. Autopsy.*

May 9, 1910. Admission of Mrs. S. S., aged 26, referred by Dr. H. G. Beck of Baltimore. The patient, whose clinical history has previously been given in full,[1] had an intrasellar struma which had burst through its capsule and extended into the cranial chamber to the left of the chiasm, which, with the tracts, was crowded far to the right side. While there had been periods of blindness dating back seven years, the condition of left homonymous hemianopsia had developed during the preceding three years (Fig. 16). She died shortly after a transphenoidal operation.

[1] Cushing, H., *The Pituitary Body and Its Disorders*, Case 2, p. 36.

At autopsy a large struma was found (Fig. 17) extending into the third ventricle and crowding the chiasm to the right. The chiasm and vessel-free nerves were available for microscopic study.

Discussion. The chiasm is shown to be much distorted (Fig. 18) by the pressure from the left side, yet the fibers on the side of pressure show less damage than those in the opposite half of the nerve, indicating that pressure alone cannot account for the atrophy which is seen. In fact, the fibers nearest the point of pressure appear to have withstood atrophy remarkably well, but it is probable that distortion of the tracts to the right with traction on the chiasm has been the damaging element. The atrophic area doubtless largely represents macular, crossed, and uncrossed fasciculi from the functionless left tract. (Fig. 18, x.) Atrophy of an uncrossed fasciculus, because of the intermingling with the crossed fibers which takes place in the chiasm, should not in any event show clearly in the chiasm, but here there is the additional difficulty of orientation of the different tracts owing to the distortion.

The sections taken from just in front of the chiasm (Figs. 19 and 20) differ considerably, the left showing a diffuse, the right a more sharply defined degeneration. The condition on the left may be due to the fact that crossed fibers representing the temporal field in the left eye have suffered as well as the uncrossed fibers. This condition would be suggested by the left field, which shows (Fig. 16) marked constriction of the temporal peripheries, and though this same thing has occurred in the other eye so that the fields are about equal for the two eyes, the left has lost considerably more in area than the right. In other words, the uncrossed fibers of the right nasal field have been best preserved, being farthest from all pressure or tension effects throughout their course. It must be noted that this phenomenon is the common occurrence in the group of homonymous cases—namely, that where the uncrossed group of fibers are affected close to the chiasm, the crossed group to the same eye are usually also affected, so that the resulting picture of atrophy in the nerve is rather diffuse.

Another striking point is the apparent decrease in the amount of atrophy of the bulbar end of the nerve as compared

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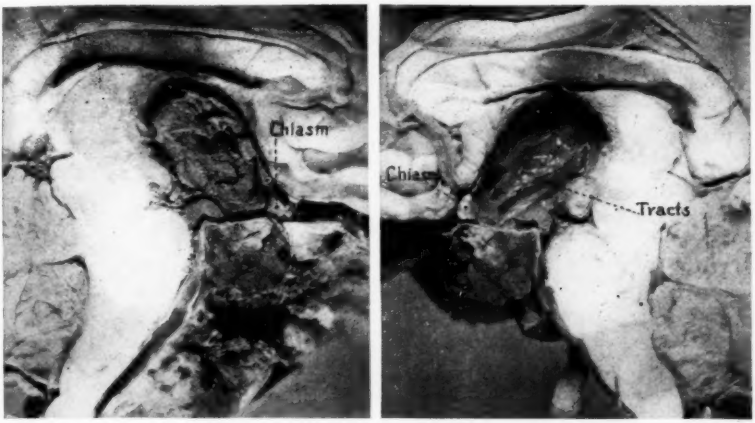

FIG. 17 (*a* and *b*).—Case III. Median section through brain. Left showing interpeduncular extension of tumor *in situ*. Right showing flattened tracts in bottom of mold from which tumor has been removed.

FIG. 18.—Case III. Section through distorted chiasm (*cf.* Fig. 17) showing chief degeneration in a crescent through X. Arrow points to side of compression.

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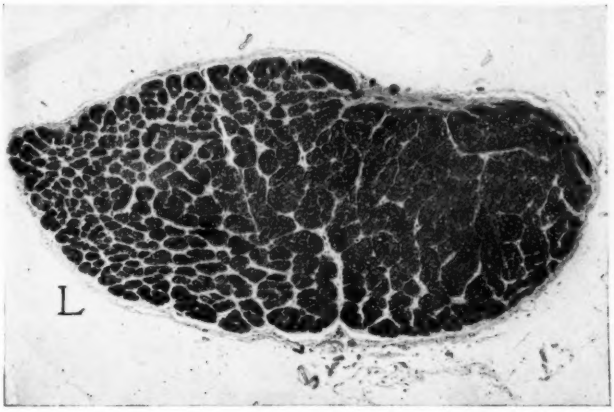

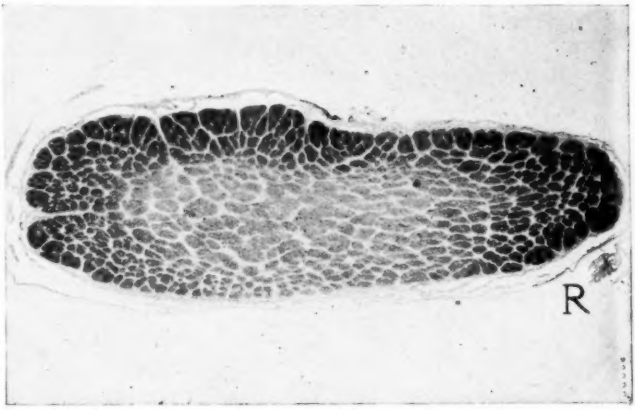

FIGS. 19 and 20.—Case III. Section of optic nerves immediately in front of chiasm (*cf.* Fig. 1, Section IV). Showing diffuse degeneration on left, particularly of the uncrossed bundles. On the right the nerve is flattened and distorted and the crossed and macular field which has swung to the bottom is highly atrophied.

with the portion near the chiasm (*cf.* Figs. 19 and 20, 21 and 22). This is best shown in the right nerve (Figs. 20 and 22) where the demarkation is sharper than in the left nerve, for the reasons mentioned. It is noticeable in both nerves, however, and may be taken to signify an ascending atrophy extending from the point of interruption toward the retina and supporting ganglion cells. If this is true, it fits well with the finding that atrophy takes place first in the tract or peripheral end of the nerve with respect to the ganglion cell. This sequence, as we shall see in other cases, is apparently modified at the vessel-bearing portion of the nerve, both by the narrowing of the nerve and consequent decrease and finally absence of medullation at the lamina cribrosa and also by the entry of the vessels taking up space and further compressing the fibers in the anterior 10*mm* of the nerve.

In the following example, one of bitemporal hemianopsia, the histological evidences of degeneration in the nerves are somewhat more pronounced on the right than left, but in both nerves a fascicular area of degeneration representing the dorsal and ventral crossed and macular fasciculus can be made out. The case was one very similar to Case 1, given in full above, though the symptoms were of longer duration.

CASE 4.—(J. H. H., Surg. No. 28818.) *Hypopituitarism; interpeduncular cyst; bitemporal hemianopsia.*

November 23, 1911. Admission of W. T. B., aged 48. Headaches and drowsiness for four or five years. Vision began to fail noticeably fourteen months ago. Diplopia for moving objects during first six months only. Increasing drowsiness with disorientation and frontal lobe symptoms. Chilliness, polydipsia, and polyuria.

Examination on Admission. Eyes: Pupils unequal, left slightly larger than right and shows less reaction to light. Both sluggish to accommodation and consensual reaction. Movements unrestricted in all directions; no nystagmus. No diplopia, exophthalmos, or lack of parallelism at present.

Fundi: O. U. disks pale, margins clear, slightly irregular. Lamina cribrosa faintly seen in the optic cup. Veins well filled, perhaps slightly tortuous. No perivascular streaking. Disk O. S. paler than in O. D., evidently some optic atrophy present.

The patient died *April 30, 1912*, after having had several

operations, so that by the time the nerve sections were obtained near blindness had been present in the left eye for a year, followed by improvement for a few months after operation, and then by total blindness for two months before death. In the right eye a fairly stationary hemianopsia (Fig. 23) persisted until the time of death.

The autopsy disclosed a typical interpeduncular cyst (Fig. 24) similar to that present in Case I.

DISCUSSION. The duration of the stage of complete bitemporal hemianopsia was long enough and the lesion sym-

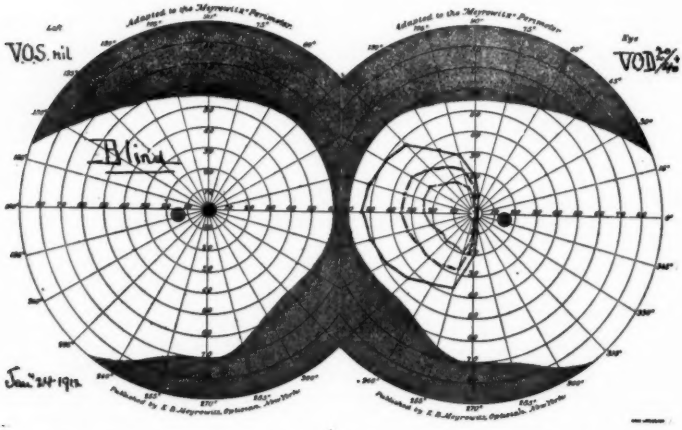

FIG. 23.—Case III. Field of Jan. 24, 1912, showing condition present before death (0.5*cm* disk).

metrical enough so that the picture of atrophy in the anterior vessel-free section of the right nerve (Fig. 26) shows an almost typical Henschen diagram with extreme atrophy of the crossed macular bundle and distinguishable atrophy also of the larger crossed fasciculi representing the temporal field.

In the left nerve (Fig. 25), blind for almost a year, the same picture of atrophy is present though somewhat more advanced. In addition to the atrophy of the crossed bundles, the uncrossed fasciculi, both macular and peripheral, are also considerably damaged. Nevertheless, the number of preserved fibers are so numerous that, could there have been relief from

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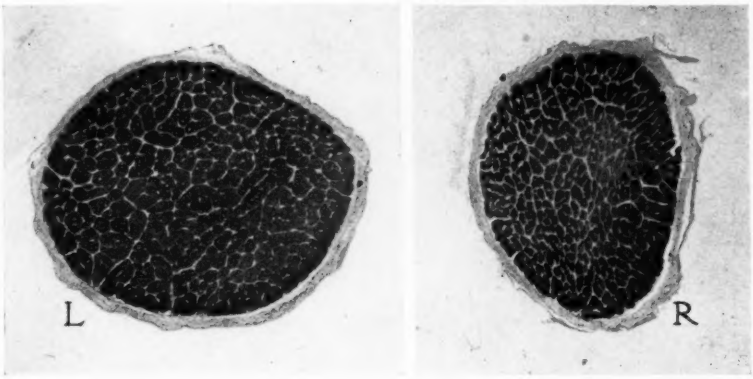

FIGS. 21 and 22.—Case III. Section of nerves in orbital vessel-free portion (*cf.* Fig. 1, Section III.), showing less marked evidence of degeneration than in the same nerves nearer the chiasm. (*Cf.* Figs. 19 and 20.)

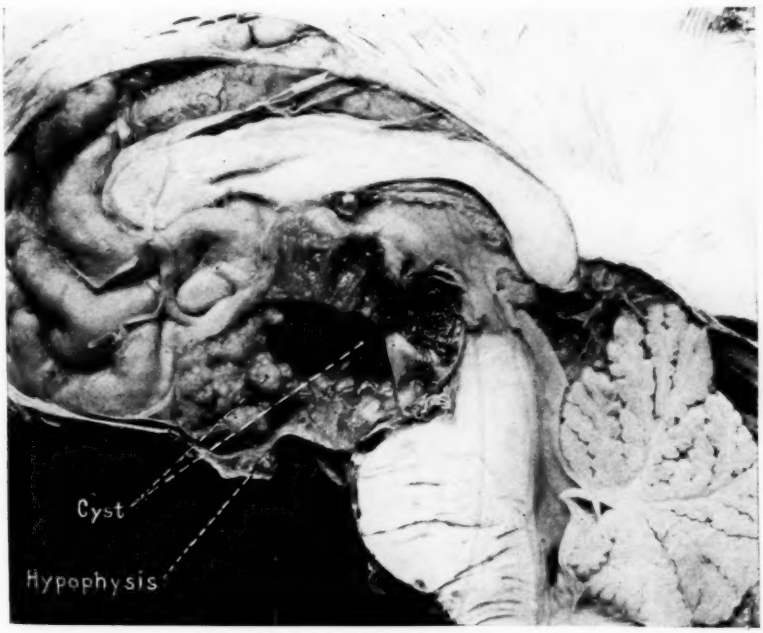

FIG. 24.—Case V. Showing interpeduncular cyst above flattened hypophysis.

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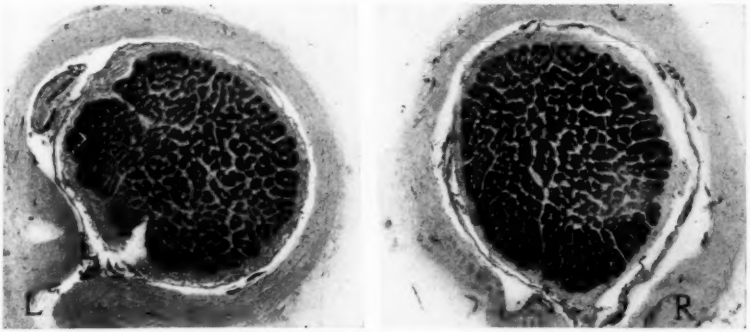

FIGS. 25 and 26.—Case IV. Section of anterior vessel-free portion of optic nerve (*cf.* Fig. 1, Section II.) showing degeneration of crossed and macular bundles.

FIG. 28.—Case V. Showing characteristic interpeduncular cyst with gelatinous contents.

pressure, the resulting vision would probably have been considerable.

The condition of the left nerve in the following case is very similar to the right nerve in the preceding one with respect to the character of the atrophy. Both represent the atrophic condition in that interesting stage where the advancing loss of the temporal field appears to be temporarily checked or to lose its impetus at the midline, although the tumor continues to grow probably at about the same rate as before.

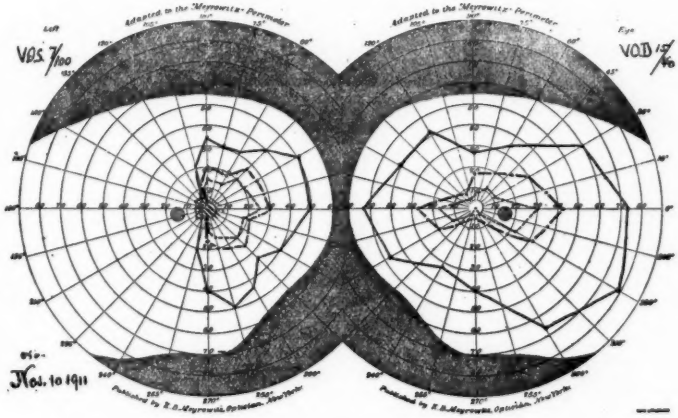

FIG. 27.—Case V. Fields of Nov. 10, 1911. Left eye in Stage IV of temporal hemianopsia (0.5*cm* disk).

In the following case the time duration was possibly twice that in the preceding case, and the atrophy correspondingly greater.

CASE 5.—(J. H. H. Surg. No. 28747.) *Infundibular cyst. November 12, 1911.* Admission of Mr. D. M. B., aged 31, with complaint of loss of vision. Bitemporal headaches for four years. Failure of temporal vision began two years ago on the left. Diplopia for four or five months. Drowsiness, loss of memory and energy, change in disposition, diminution of libido, etc., have been noted. Sella slightly enlarged. Floor thinned.

Examination of Eyes. No exophthalmos, diplopia, or unbalance in extrinsic muscles. Right pupil slightly larger

than left. *Fundi* in good condition, except for some increase in disk pallor, which is more marked in O. D. Fields (Fig. 27) show temporal hemianopsia in O. S. and slightly contracted fields in O. D. V. O. S. $\frac{7}{100}$; V. O. D. $\frac{15}{40}$.

November 12, 1911. Operation. Sellar decompression, followed by meningitis and death on *December 3d.*

The autopsy revealed an interpeduncular cyst with gelatinous contents (Fig. 28).

Comment. With a temporal hemianopsia of three or four years' duration in the left eye and with a central scotoma, it is not surprising that the nerve on that side shows (Fig. 29) a well-marked atrophy of the macular and crossed fibers with some damage to the uncrossed group, as contraction of the nasal field and enlargement of the central scotoma took place. This indicates, as does the previous case, that when a temporal hemianopsia remains fairly stationary for a year or more, the atrophy found in the nerve will be quite definite, although a little magnification shows that the atrophic areas are still abundantly supplied with undegenerated fibers.

The degree of atrophy in the right nerve (Fig. 30) is very slight and the cyst must have pressed against the right tract implicating the crossed bundle from the left eye.

The following case is an example of a still more advanced condition.

CASE 6.—(J. H. H. Surg. No. 29150.) *Malignant hypophysial adenoma with interpeduncular cyst. Marked neighborhood and general pressure symptoms. Operation. Death. Autopsy.*

January 25, 1912. Admission of Mr. I. B. A., aged 33. Patient returns after having had a subtemporal decompression seven months ago for relief of pressure symptoms and failing vision which began three years ago. Six months ago V. O. D. became nil. Recently (two or three months) patient has developed a left-sided weakness and incontinence of urine in addition to aggravation of all previous symptoms. X-ray shows enlarged sella. Fields are shown in Fig. 31.

Eyes. Pupils about equal and small; no direct or indirect pupillary reaction to light. No nystagmus. Movements of eyes rather jerky. Apparently some weakness of third and fourth nerves on the right. *Fundi*: Disks very pale with sharp, somewhat irregular margins. Lamina cribrosa and

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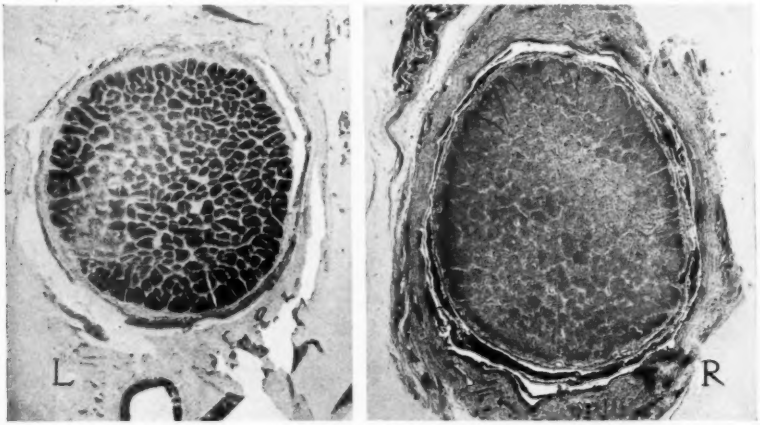

FIGS. 29 and 30.—Case V. Section of optic nerves in vessel-free segment (*cf.* Fig. 1, Section II). Right nerve well preserved. Left nerve with characteristic atrophy of crossed bundles.

FIG. 32.—Case VI. Section of brain showing malignant adenoma probably originating from the large superimposed and presumably congenital cyst.

optic cup obscured by a layer of new tissue. Arteries small. Veins not overfilled and slightly tortuous. Slight amount of old scar tissue on large vascular trunks.

February 7, 1912. Transphenoidal operation. No relief from symptoms and death occurred on *February 14, 1912.*

The autopsy showed (Fig. 32) a malignant tumor of the gland with a superimposed cyst.

Comment. In this case the nerves after three years of pressure in the chiasmal region and tracts show decided degeneration, particularly in the right nerve, which had been

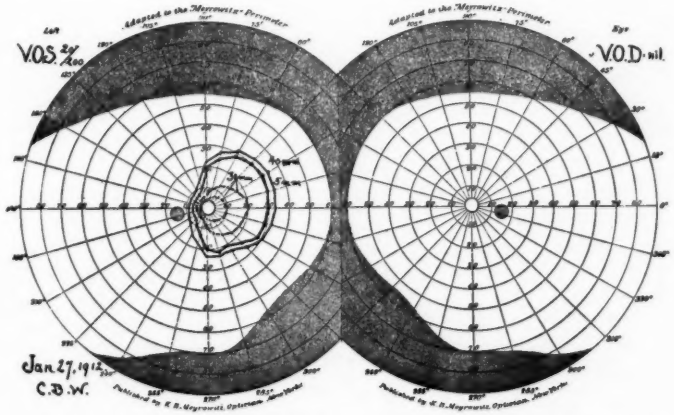

FIG. 31.—Case VI. Fields of Jan. 27, 1912. (Inner form field = 0.5*cm* disk; outer = 4*cm* disk.)

totally blind for six months. In the left nerve well-marked degeneration, especially in the crossed bundles, can be made out and the atrophy evidently follows the paths indicated by Henschen. (Figs. 33–40.) While the crossed macular and field bundles in this left eye show the greater atrophy, the uncrossed tract has also been considerably affected, but the macular bundle is unusually well preserved for such an advanced case. Indeed, the vision of $\frac{20}{200}$ is exceptionally good for this stage of hemianopsia.

It would be difficult to tell from the condition of the right nerve whether the process began as a nasal or temporal defect, but for the most part the uncrossed tracts seem somewhat

best preserved, so that very possibly the case began homonymously.

The cases so far serve to give an idea of how much atrophy may be expected after a duration of pressure in the chiasmal region of from six months to two or three years. In the atrophic areas of the nerves, even in the long-standing cases, it is notable that great numbers of fibers may be preserved after a pressure block in the chiasmal region. Conceiving that this preservation was largely accomplished by support from the retinal ganglion cells, it became interesting to note how long such support could maintain the axone in the optic nerve. The process in the following case was an advanced one, the patient having been completely blind in the right eye for six years and in the left eye for three years with but the faintest light perception for five years.

CASE 7.—(P. B. B. H. Surg. No. 1784.) *Acromegalic gigantism. Large adenoma with intracranial extension.*

September 17, 1914. Admission of M. van W.,[1] age 41, referred by Dr. Marxmiller of Los Angeles. At the age of 28 he began to have severe headaches with acromegalic overgrowth. The vision began to fail in the left eye in 1905 and was finally lost early in 1910. By the end of that year, when he first came under observation, the right eye also had become nearly blind. Both eyes showed the intense pallor of optic atrophy and only two small patches of vision could be plotted with large disks in the lower field of the right eye.

On *December 17, 1910*, a transphenoidal operation was performed with removal of some of the tumor from the enormously enlarged sella. This was followed by a lessening of discomforts and widening of the seeing field, which assumed the character of a superior hemianopsia.

After a period of improvement vision was gradually lost. He now returns after four years for further help owing to an exacerbation of the headaches.

Examination of Eyes. The globes are large, protruding, and show a divergent squint. The disks are extremely pale. Total blindness for three years. No pupillary reactions.

Attempts were made to relieve his discomforts, which were evidently due to an intracranial invasion of the growth, by a subtemporal decompression and later by a third tran-

[1] The earlier history of this patient has been recorded: *cf. The Pituitary Body and Its Disorders*, Case 1, p. 30.

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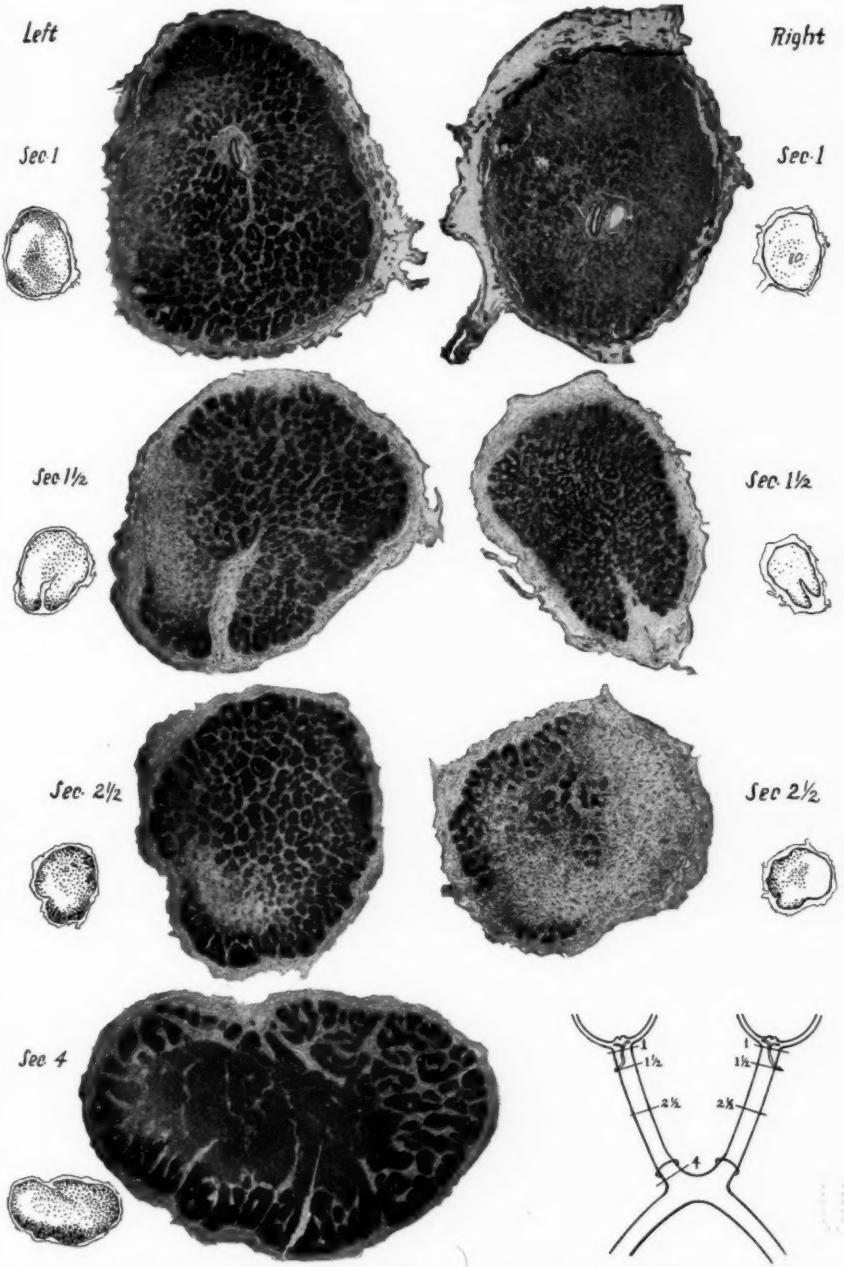

FIGS. 33–40.—Case XI. Showing sections of nerves at level indicated in diagram. Diffuse degeneration on the right, chiefly in uncrossed fasciculi; on the left, degeneration less diffuse and chiefly confined to crossed fasciculi. Condition therefore probably caused an original homonymous hemianopsia. Compare small sketches in which best-preserved bundles are represented in dots with Henschen sectional areas (Fig. 1).

sphenoidal operation, but they were unavailing and after five months he succumbed.

Autopsy. February 16, 1915. A large intracranial extension of a pituitary adenoma was disclosed (Fig. 41), the tumor having crowded the tracts and chiasm to the right, leaving a mere ribbon of tissue.

Discussion. After such a long period of blindness one would have expected to find in this case, if in any, a complete degeneration of the nerves, and though the microscopic sections (Figs. 42 and 43) show an advanced state of ascending atrophy, nevertheless there are many normal fibers still to be detected even with a low power of magnification. The preserved fibers are most numerous in the peripheral region of the uncrossed fasciculi of the right eye, but even on the left, blind for six years, there are numerous intact fibers, as are shown under higher magnification in the drawing (Fig. 44).

In the following and final case, even though there was total blindness and the nerves were actually compromised by extension of the tumor into the sheath of Schwalbe, some normal fibers were still retained in the nerves.

Case 8.—(J. H. H. Surg. No. 28200.) *Large infundibular endothelioma of long duration. Neighborhood and general pressure symptoms of advanced degree. Hypopituitarism. Terminal medullary phenomena. Subtemporal decompression without avail.*

August 16, 1911. Admission of Mr. F. H.,[1] age 41, referred by Dr. Whoriskey of Cambridge, Mass. Symptoms of pituitary insufficiency for several years. Severe headaches for five years with rapidly failing vision. In 1907 a bitemporal hemianopsia was found with near blindness (Stage VII) on the right, and about two years before admission he became completely blind. Marked stupor present. Total anosmia.

Examination of Eyes. Marked bilateral exophthalmos, more pronounced on right than left; positive Von Graefe; bilateral optic atrophy; *fundi* otherwise negative; no papilledema despite extracranial circulatory stasis with passive congestion and œdema of lids and conjunctiva. Ocular movements limited and elicit nystagmoid twitches both to right and left. Slight divergent squint on right. Constant

[1] Previously recorded from another standpoint: *cf. The Pituitary Body and Its Disorders*, Case 8, p. 63.

oscillatory movements. Pupils small and equal. No reaction.

Death occurred on the third day after a subtemporal decompression.

The autopsy revealed an enormous suprasellar endothelioma invading the ethmoid region (Fig. 45). Pituitary body flattened to thin crescent. The intracranial course of the optic nerves was not determinable, but sections of the intraorbital portion (Fig. 46) show that the growth has extended down into the sheath.

DISCUSSION. Here, as in the preceding case, normal fibers are still demonstrable and appear to be chiefly distributed in the uncrossed fasciculi, but this is not entirely clear. At all events, could the nerves have conceivably been relieved of pressure, some light transmission probably in the temporal fields could have been regained.

This series of observations makes it apparent:

(*1*) *that the anatomical changes in the extracerebral pathway in cases of pressure against tract, chiasm, or nerve are far less sharply cut than the functional disturbances determinable by the perimeter would lead one to expect;*

(*2*) *that even in cases of hemianopsia with a vertical meridian and a hemianopic pupillary reaction of several months' duration the crossed bundles may show only a scattering of degenerated fibers indicating that the block is largely a physiological one;*

(*3*) *that in cases of longer duration areas of degeneration, more marked on the cerebral than on the orbital side of the point of pressure, begin to show on sections and correspond with considerable definiteness to the fascicular distribution described by Henschen;*

(*4*) *that even in long-standing cases of total blindness with extreme pallor of the nerve-head and loss of pupillary reaction, intact fibers are still histologically demonstrable on the retinal side of the point of pressure.*

It is our impression that the diffuse picture of degeneration in the nerves is due to the gradual progress of the pressure as indicated by the advancing changes in the fields which we have spoken of (8) as Stages of Hemianopsia. It is probable that a case which has reached Stage IV—that is, the stage with a vertical division, and which remains at the stage without further progress for six months or over—would show a sharply

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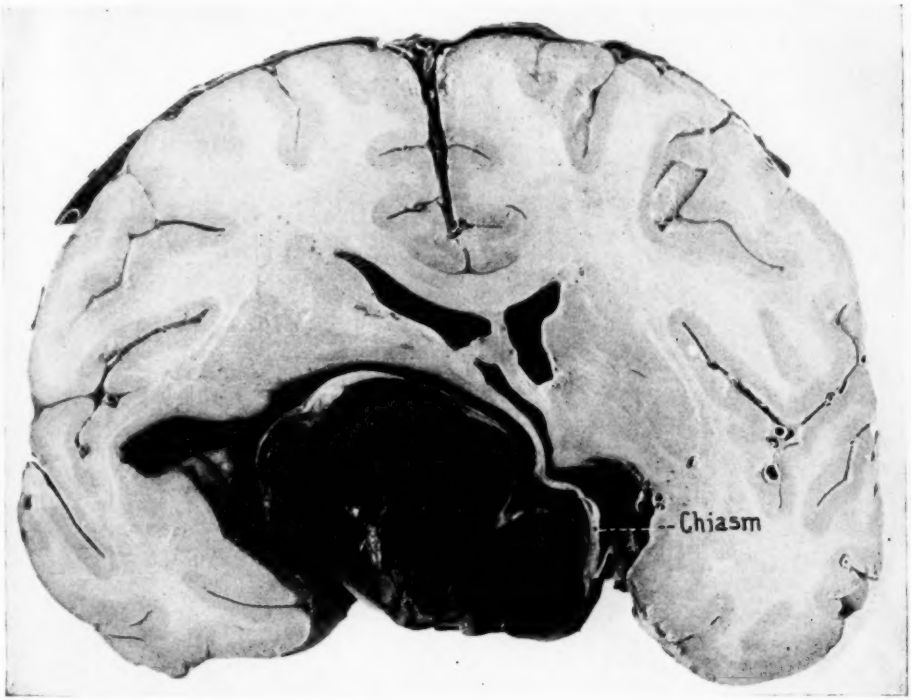
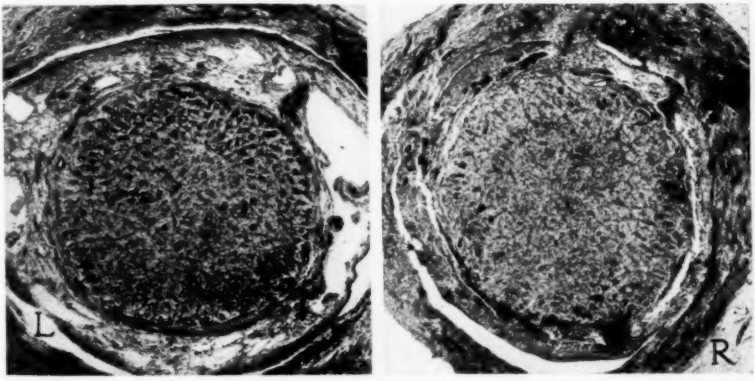


FIG. 41.—Case VIII. Coronal section of brain viewed from behind, showing the intracranial portion of the tumor and chiasm displaced to the right.



FIGS. 42 and 43.—Case VII. Sections of vessel-free portion of optic nerves showing extreme sclerosis with preservation of new fibres.

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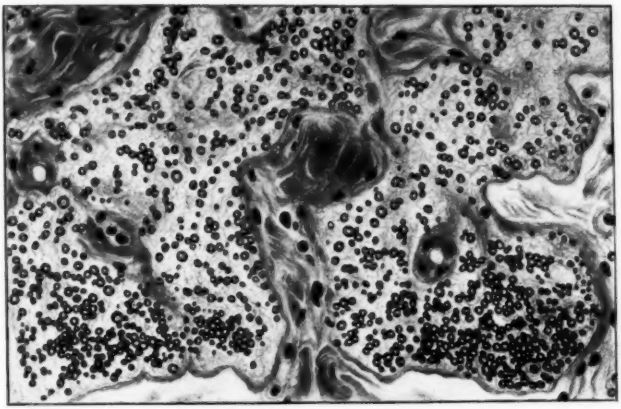

FIG. 44.—Case VII. Field from left nerve (*cf.* Fig. 42) under higher magnification (x 500) showing intact fibres.

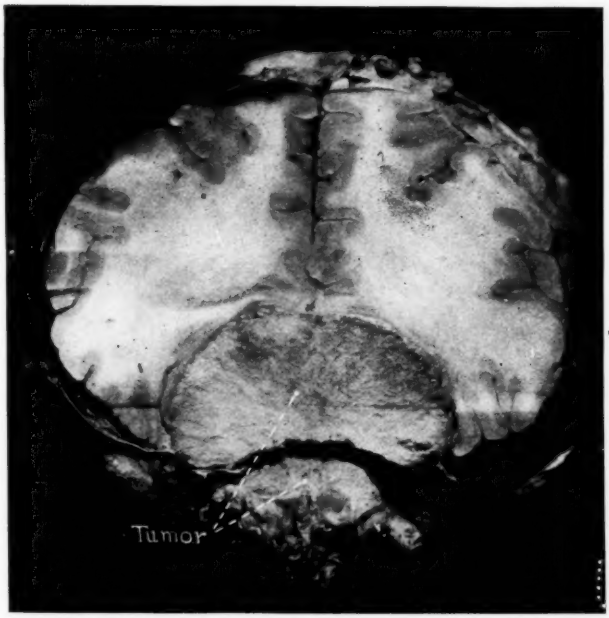

FIG. 45.—Case VIII. Coronal section through tumor. Position of chiasm and tracts not determinable.

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FIG. 46.—Case VIII. Showing highly atrophied nerve with extension of intracranial endothelioma into optic sheath. Process bilateral. Some intact fibres still demonstrable.

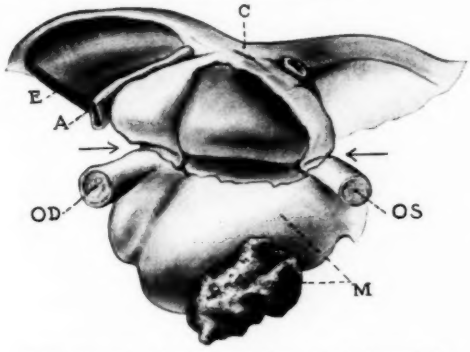

FIG. 47.—Showing from in front the chiasmal distortion produced by an hypophysial adenoma with intracranial extension. M = the intrasellar tumor mass, C the chiasm, OS and OD the optic nerves which have been cut at the level of the arrow by the band of dura which connected the two anterior and widely separated clinoid processes. An artery (A) has also deeply cut the right nerve near the chiasm where it overlies an extension of the tumor (E) out into the right temporal lobe.

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cut fascicular degeneration. The nearest example of this is in the right eye of Case 4 (Fig. 26). As a matter of fact, there are many elements which enter into the chiasmal distortion which may affect the character of the fields and which are not thoroughly understood. This applies particularly to cases in which a tumor originating in the gland has finally burst through the capsule and invaded the cranial cavity. Under these circumstances the chiasm is lifted by the growth, the intracranial portion of the nerves greatly elongated and flattened and they may be seriously cut or strangulated by adjacent arteries or by the interclinoidal margin of dura. In Fig. 47 is shown a preparation of such a tumor and chiasm from which the brain hardened ***in situ*** before removal has been carefully dissected away. The patient showed a type of left homonymous hemianopsia with an irregular involvement of the right temporal field, and the conditions found after death show how many factors in advanced cases may come to affect the nerves and consequently the fields.

III. PREVIOUS OBSERVATIONS

(*a*) *Pathological.* Having thus been led to conclude that a ganglion cell of the retina can support its central neurone up to the point of pressure in the chiasmal region long after such pressure had produced functional discontinuity and even atrophy of that part of the neurone central to the point of pressure, the observations of our predecessors bearing on this matter should be recorded.

Henschen (18) gathered seventeen cases in which known visual defects were followed by satisfactory pathological examinations; eight of these were his own and the rest taken from the literature. Toxic central scotomata, blindness in one eye only, and homonymous hemianopsias were represented in the collected series, but there were no examples of hemianopsia due to lesions in the chiasmal region except one case, a gummatous lesion of the tract of six months' duration which showed a very slight amount of nerve degeneration. In the other cases with hemianopsias and scotomata the time duration was longer, but even in the tracts pointed out as degenerated after one to fifteen years, it must be noted that the areas were well

filled with preserved fibers, although by contrast other areas were better preserved. However, these areas in serial section were definite enough so that a very sharply defined diagram was made and has stood the test of time remarkably well.

Not a great deal has been since added from a pathological standpoint to these tract diagrams of Henschen's, although many reports have been made on optic nerve sections (1) in cases of toxic scotoma and (2) after the enucleation of one eye. Of cases of posterior origin (3) with homonymous hemianopsia there have been numerous reports, but those with hemianopsia due to hypophysial or parahypophysial tumors have been very few.

From the first (scotoma) group the position and semi-decussation in the chiasm of the papillo-macular fibers has been fairly definitely determined.

In the second group of cases (monocular blindness) the partial decussation in the chiasm and the course in the tracts and primary centers can be studied. In this group the nerve stump usually shows many preserved fibers increasing in number toward the chiasm, which may represent either commissural or centripetal fibers from the higher centers or both. A few cases having portions of the retina destroyed by tumor disease or injury have also been useful in determining tract relations.

In the third group, comprising the homonymous hemianopsias of central origin, both the crossed and uncrossed paths may be studied if sufficient time has elapsed for degeneration to occur in the retino-geniculate part of the pathway. This may be demonstrable within a year, but for as long as fifteen years there may still remain large numbers of intact fibers in the degenerated areas. Thus Schmidt-Rimpler (28) reports a case of homonymous hemianopsia of two years' duration resulting from a traumatic cerebral cyst in which the optic nerves showed no atrophy, and even in a case of five years' duration the atrophy was very poorly defined. This gives an indication of the slow occurrence of secondary atrophy in the anterior neurones after lesions of the geniculo-calcarine portion of the pathway.

Of the fourth group of cases, with which we are chiefly concerned, with the lesion near the chiasm, the reports have

been few. Dimmer (13) and Sachs (25) have recorded three examples with which some of our findings accord—namely, a diffuse atrophy in the nerves although the tracts were reduced to mere ribbons.

(*b*) *Experimental.* On the experimental side of this question, much has been done on various animals from frogs to monkeys. For the most part the procedure has been to make lesions in the retina, the nerve, the chiasma, the tracts, or the primary centers, and to study the degeneration by serial sections and Marchi stain after three weeks to six months have elapsed.

The retinal sections have yielded striking results with comparatively little operative labor. Although Falchi (14) and Baquis (3) first made retinal sections to study the effect on the retinal elements themselves, it was not till a decade later (1895) that Pick (23) used the method on rabbits to study degeneration in the nerve and tracts. A cataract knife was simply pushed through the sclera at the ora serrata and under observations with the ophthalmoscope a small cut was made in the retina down to the sclera close to the optic disk. Sections were made in all retinal quadrants and always a corresponding degeneration in the quadrant of the nerve could be traced unaltered in position throughout its length and on through the chiasm into the tracts. However, as Henschen states, these findings do not necessarily prove that the fibers in man have the same arrangement.

In 1896, Dean and Usher (12) without knowledge of Pick's work performed similar experiments on monkeys with the same results. After section of the optic nerve they found that atrophy extended both ways, but more rapidly toward the brain. They furthermore used the electrocautery to destroy the papillo-macular bundle and found its course to be as Henschen and others had outlined. Pick, Dean, and Usher all found in addition to the definite quadrant of atrophy not only scattered atrophic fibers on the opposite quadrants but also scattered atrophic fibers in the nerve of the other side belonging to the uninjured eye. Parsons (22) in 1902, being skeptical as to these latter findings, repeated the experiments on monkeys and was forced to agree with the finding of scattered atrophy in both nerves when only one was injured. Such a phenome-

non may argue for the presence of commissural fibers or possibly for a branching of fibers as Ramon y Cajal (9) has demonstrated microscopically.

Many sections of the optic nerve both in the orbit and intracranially have been made for various purposes. Wagemann (29) found that after section of the optic nerve in the orbit the result varied according to the amount of vascularization disturbed. If the central vessels of the nerve were cut there was rapid degeneration of the bulb end of the nerve and also considerable retinal disturbance—the latter depending greatly upon the number of ciliary arteries cut. If the section was farther back so that the central artery was not damaged, and the ciliary vessels disturbed as slightly as possible, then the atrophy took place very slowly toward the eye as compared with the central stump, so that after six months a great many fibers in the bulb end still remained intact. The length of time these fibers may survive has not been determined experimentally.

With regard to the effects of an intracranial section of the nerve without orbital vascular disturbance or trauma, there is considerable disagreement. Krenchel (19) and Hamburger (17) found in frogs no atrophy of the retinal ganglion cells or intraorbital portion of the nerve after six months except a millimeter or so from the section, whereas Lehmann (20) and Lent (21) after similar sections in frogs and dogs observed a marked atrophy. Birsch-Hirschfeld (5) claims to have noted minute changes in the retinal ganglion cells by the delicate Nissel method in fifty-five hours after section.

Numerous observers have made sections of the chiasm and tracts both to study the pupillary reaction and the optic paths. While most of these observers studying degeneration have been able to obtain enough degenerate fibers to locate tracts, some have found only a diffuse weak secondary atrophy.

IV. GENERAL DISCUSSION

The eight cases which have been presented will suffice to give some idea of the occurrence and progress of atrophy in the optic nerves following lesions affecting the chiasmal region. So far as the study of the position of the fasciculi in man is

concerned, little can be gained by animal experimentation, nor are acute experimental lesions comparable with the chronic ones we are considering. Thus, when sections are made in the optic nerve or tract of animals, degeneration has been shown to take place in both directions with equal step. Again, retinal section, even in the monkey, has failed to give more than an approximate idea of the paths in the optic nerve except as regards its papillo-macular bundle, which is disposed similarly to that in man. Certainly Henschen was quite justified in insisting that the position of the fasciculi be worked out on human material; and so far as our few observations go the bundles are distributed in practically the situations which he described.

From our observations, too, it seems probable that the degeneration occurs primarily in the tracts, where it can be observed by Weigert stains in about six months after functional block of vision has taken place and before degeneration appears in the optic nerve itself. When the optic nerve becomes affected the vessel-bearing portion, where the nerve begins to narrow to enter the lamina cribrosa, appears to show a more advanced degeneration than the vessel-free portion (*cf.* Case 1). Here each individual fiber must have a relatively smaller amount of myelin surrounding it than is present farther back, and the decrease in size is augmented by the entry of the central artery with its vein or veins and fibrous support. Or the condition may represent an early peripheral atrophy of the centrifugally running fibers exactly as the initial atrophy in the tracts represents the peripheral atrophy of the centripetally running fibers, as Sachs (25) has suggested. The atrophy in the nerve may thus be regarded as a simple atrophy, both ascending and descending, secondary to chiasmal distortion whether this be due to pressure or tension.

This corresponds with the clinical appearance of the disk in the first six months of the onset when its pallor is usually not outside of possible physiological variations. As the pallor does make a definite appearance, it is usually first notable in the temporal side at the point of emergence of the papillo-macular bundle, which microscopically also shows severest damage. The pallor of the nasal portion representing crossed fibers is usually masked by the normally high color of that

region until a much later period, so that there is rarely found a notable contrast between the areas of the disk for crossed and uncrossed fibers.

The ophthalmoscopic picture of this form of atrophy may in certain stages closely simulate the atrophy of toxic origin, but rarely is the excavation of the cup or the sharpness of the lamina cribrosa as great in the former as in the latter. Indeed, one may find some blurring of these details by a light layer of tissue resembling the secondary changes following a low grade of papillœdema. But as a matter of fact it is remarkable that, despite the large intracranial extension of the tumors in question, there is rarely any evidence of what may be called a choked disk and its absence is due in all probability to the fact that there is rarely any increase in intracranial tension in these cases, and when it does occur in terminal conditions as in Cases 7 and 8 the optic sheath has become so occluded that cerebrospinal fluid cannot be forced down into it and lead to œdema.

After the first six months the atrophy in the nerve becomes more or less well defined with the persistence or advance of the pressure. If the growth of the tumor is rather slow and symmetrical, as in Case 4, where the stage of more or less complete bitemporal hemianopsia was held for six months or so before the uncrossed tracts were much affected, the atrophy of the crossed bundles and macular fibers will give the characteristic picture as described by Henschen. If, however, the growth is more asymmetrical or more rapid, the damage to one group of fibers is not complete before another group is affected, thus blurring the contrast picture of fascicular degeneration in the nerve. In this respect the degenerations following chiasmal lesions differ markedly from lesions farther back where the damage will remain confined to one or two fascicular groups, producing after six months or a year well-defined atrophic areas in the nerves. Thus the chiasmal region is comparable to the focal point of a lens system in that relatively small obstructions here produce relatively magnified results, so that even small variations of the obstructions are notable in the field. The atrophies consequently are apt to be more diffuse than those from cerebral injuries. This is clinically well shown by the phenomena of response to large disks

so often seen in these anterior lesions and not in posterior lesions.

It has seemed to us also that the atrophy in the nerve decreased from the chiasm to its vessel-bearing portion where other modifying factors may enter in. This phenomenon can be explained as the result of a retrograde atrophy. Moreover, the crossed bundles appear histologically to give sharper atrophies than do the uncrossed bundles (*e.g.*, Figs. 19–20, 21–22). This may be attributed to a combination of such factors as the anchorage due to the interweaving of crossed fibers in the chiasm and their consequent inability as compared with the uncrossed fibers to recede when under pressure without their being subjected to tension. It can hardly be due to any absolute difference in susceptibility of crossed and uncrossed bundles, because posterior lesions are apt to affect both sets alike.

CONCLUSIONS

1. Despite the so-called atrophic pallor of the disks in patients having visual-field defects resulting from lesions in the chiasmal regions, the histological examination of the nerves fails to show the expected degree of fiber degeneration unless the process has been of long duration.

2. The atrophy in the tracts considerably antedates that in the nerves, where the fibers may be preserved by their retinal ganglion cells for several years after complete functional blindness has occurred.

3. Our cases serve to illustrate the fact that in the presence of chiasmal pressure of known long duration associated with sharply cut hemianopsias, even when to the ophthalmoscope the nerve shows the pallor of presumed atrophy, there may be no corresponding sharp delimination of the areas of atrophy in the cross-sections of the nerve.

4. This at first sight would appear to be an inconsistency, but our more accurate perimetric findings with graded disks show that, after all, the boundaries of the seeing areas are less sharply cut than we had previously supposed, and perhaps correspond after all with the diffuse picture in the nerves.

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ON THE DECLINATIONS OF THE VERTICAL MERIDIANS OF THE RETINA.

By GEORGE T. STEVENS, M.D., Ph.D., New York.

(With six figures in the text.)

Second Paper

IN the former paper I have stated that the heterophoric conditions, esophoria, exophoria, and hyperphoria, are in reality manifestations of declination, and that the rational, effectual correction of heterophoria depends upon the correction of declination. The same principle extends to the correction of heterotropia or strabismus.

If the general proposition is correct it follows that, in either the deviating tendencies of heterophoria or the actual deviations of strabismus, it is the cause of the tendency or of the deviation that demands attention, and that if the cause is effectually removed neither the tendency nor the deviation should continue.

With this principle in mind it will be interesting to inquire what special conditions of declination we may expect to discover associated with special forms of heterophoria or heterotropia. At the first suggestion of a relation of cause and effect between the two conditions we might conclude that the special conditions of the one should invariably predetermine not only the form but the degree of the other.

Unfortunately, owing to the complication of forces involved in the proposition, this conclusion is correct only as a general principle while in detail there may be apparent exceptions to the rule. Such exceptions are, in fact, apparent, not real. They are due to the complications of forces to which reference

has just been made. As a practical fact the conditions are in the great majority of cases in harmony and the exceptions are not very common.

In my work on the *Motor Apparatus of the Eyes* I laid out what I termed an *empirical scheme for some of the relations of declination and heterophoria.* The scheme was offered as a tentative one, *suggestive* rather than *absolute.* Ten years of practical experience added to those which preceded this suggested scheme have so far confirmed its general correctness that I regard it as fully worthy of being included in this discussion and of the declaration that in principle the scheme is correct and also that exceptions are, as I have stated above, apparent rather than real.

Perhaps a single example may illustrate the principle:

Within a few days I have examined a physician who has for years suffered from severe asthenopia. He uses glasses which are perfectly adjusted to his refractive condition but they do not enable him to read even for a few minutes. He has manifest declination; Right + 1, Left + 5. There is *no form of heterophoria* although he was using, when I first saw him, prisms for exophoria. It is probable that he had not shown exophoria even at the time when the prisms were prescribed.

Now, as a result of so considerable declination we would expect, if our rule is correct, an important degree of heterophoria, while in fact there is practical orthophoria. Why does the rule not hold good?

In the left eye, which is somewhat amblyopic, there is the very high degree of declination. By reason of the amblyopia he is able mentally to exclude, in distant vision, a recognition of the left eye image to such an extent that he makes little or no effort to force the vertical position of the retinal image; and hence little or no effort for adjustment which would result in heterophoria is made. In looking at near objects, however, he is less able to ignore the eye with the greater defect, and not only some exophoria in accommodation results but the images of the two eyes are confused by a partial crossing of the lines of the type. In the letter *l* or *t*, *h* or *b*, etc., the long vertical lines would cross thus: ×. Confusion and fatigue would be the natural result. If space permitted, many forms of such apparent exceptions to the rules might be illustrated.

Accepting, then, the rules as in principle correct but subject to such exceptions as may be easily suggested where the mechanism is influenced by so many physical as well as mental modifications, we may represent diagramatically the results of declination in relation to heterophoria in the following paragraphs.

As the conditions of declination resulting in exophoria are perhaps those most easy to explain, let us begin with those conditions.

In exophoria there is, as a rule to which there are few exceptions, positive (+) declination for each eye and, for most cases,

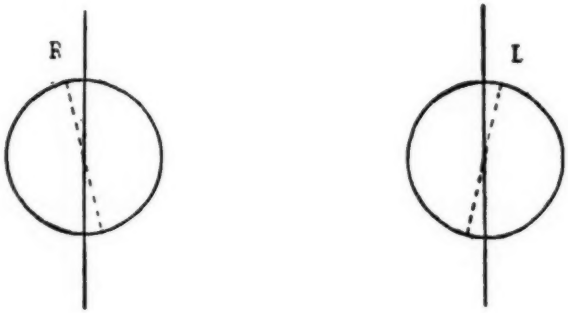

FIG. 4.—Leanings of the Meridians in Exophoria. If they are of markedly different degree we may have hyperphoria.

an approximately equal degree of leaning for the two eyes. This we may represent by the diagram, Fig. 4. The more extended lines represent the technically vertical direction of the eyes in question. The dot lines indicate the actual position of the apparent vertical meridians of the eyes. It is seen that the apparent vertical meridian of each eye leans *out* above.

In the act of vision, in order to unite images, these two obliquely turned meridians must be brought to the true vertical position or to that approximation which would be consistent with the law of Listing.

To accomplish this, certain muscles of each eye must be brought into action. In this case one of the principal forces to be put into requisition would be the superior oblique of

each eye. Assuming that this muscle (we may for the moment leave out of consideration all forces except this) were to act strongly, each of the eyes would rotate on its antero-posterior axis, it would also rotate *downward* and *outward*. The result would be, unless the action of the internal rectus of each eye should be brought forcibly into action and other muscles to adjust against the depressive action of the obliques, exophoria with depression of the plane of vision of each eye. If the declination should be equal or very nearly equal for the two eyes, there would therefore result simple exophoria, while should there be a material difference in the degree of declination there might also occur, because of the difference in the depressive action of the two superior obliques, hyperphoria. Thus we would, as the result of a positive declination of both eyes, expect exophoria; and if the declination in the two eyes should be materially different in degree, we would expect hyperexophoria.

It is not to be assumed that the oblique muscles would alone act in such a case but, for simplicity in explanation, I have mentioned only the action of these. The other forces which might be brought into action would only emphasize the results mentioned.

We may now consider another combination of leanings of the meridians from which quite a different result is to be expected. Let us now suppose the leanings to be conjugate—that is, that the vertical meridian of each eye leans to the left or to the right, one toward the medial, the other toward the temporal side. Here also the result will be modified by the relative degree of the individual leaning. If the leaning of one eye is very slight, the other considerably greater, we may expect exophoria. But with important conjugate leanings we would also expect hyperphoria.

At Fig. 5 we observe the conjugate leanings of the meridians. In order to eliminate these leanings, or to modify as far as possible the tilting, a different group of forces must be brought into exercise in each eye. In the case of a positive leaning of the left eye, for example, we would have action similar to that already mentioned in the case of the leanings out for each eye. But we must take a somewhat more inclusive view of the forces which will be brought into action for each eye.

For the right eye, then, we shall have the action, first of the inferior oblique which will rotate the eye up and out and roll it to the temporal side. To neutralize the upward tendency, the inferior rectus will rotate the eye down and tilt it slightly to the right. At the same time the superior oblique will act upon the left eye accompanied by an action of the superior rectus. If the declination is equal or nearly so in the two eyes, there will be hyperphoria (unless under a strong muscular impulse the tendency is resisted), but if the minus (—) declination of the right is much less than the positive (+) declination of the left, there will be, unless forcibly corrected, esophoria.

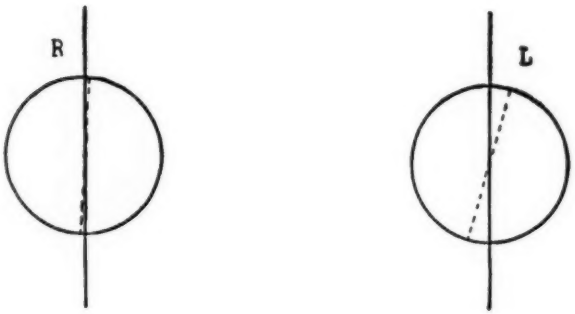

FIG. 5.—Leanings of the Meridians in Esophoria. If the leanings are both pronounced, we may have hyperphoria.

This result might appear to be unexpected, as it would be were the eyes governed only by unconscious mechanical forces. In the actual state of conscious adjustments, the effort to rotate the left eye medianward is accompanied by a synergic action of the correspondingly acting force of the right eye, with the result that it rotates in and being unrestrained, or less restrained than the other, the result is generally esophoria.

A single other combination will suffice to illustrate the principle which we have in view. Other combinations are discussed in the work on *The Motor Apparatus of the Eyes*, but we may consider the three which we are here introducing as suggestive of others.

Let us, therefore, next consider the result in case there is

an absence of declination of one eye while the other tilts to a marked degree as it is shown at Fig. 6.

In this case the muscles involved will be the superior oblique, rotating the left eye down and out and rolling the upper extremity of the meridian to the right. The superior rectus will act in compensation, rotating the left eye upward and slightly in and rolling the eye somewhat to the right. The result to be expected is esophoria and, if the right superior rectus acts synergically with the left, right hyperphoria.

Cases occur, more frequently perhaps with high degrees of declination than otherwise, where the subject of declination of

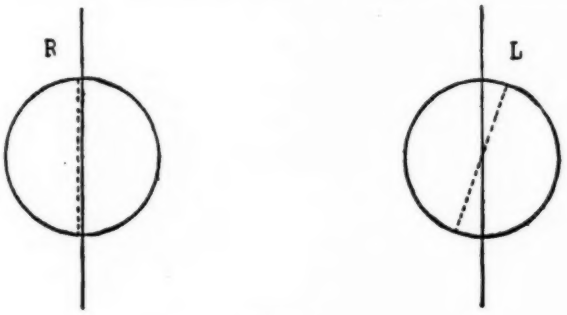

FIG. 6.

one eye principally selects to submit to the greater or less confusion from declination rather than to the effort of correcting it even partially, and there results, generally, more or less amblyopia of the leaning eye with very little or no heterophoric manifestation.

These examples should serve to strongly suggest that the conditions of heterophoria or heterotropia are corrected neither by prisms nor by reducing the force of a rectus muscle or by shortening and thus increasing the action of another.

It is a principle of greatest consequence that the conditions which give rise to heterophoria or to strabismus can not be properly corrected either by tenotomies or by contractions of any of the recti muscles.

Strabismus and heterophoria are secondary conditions, rarely if ever, except in paralysis or spasm, due to weakness or

contraction or to too great or too little length of the recti muscles. In fact ***non-paralytic strabismus is not the result of undue organic shortening or lengthening of any ocular muscle.***

As to spasm, while as a result of certain cerebral diseases it may occur, it is not of considerable moment in a discussion of heterophoria under ordinary or normal conditions, and the fact that there may be diplopia does not of itself indicate paralysis. The rotations, as indicated by the tropometer, must confirm this if paralysis is to be accepted.

From what has been said it might be thought that in all

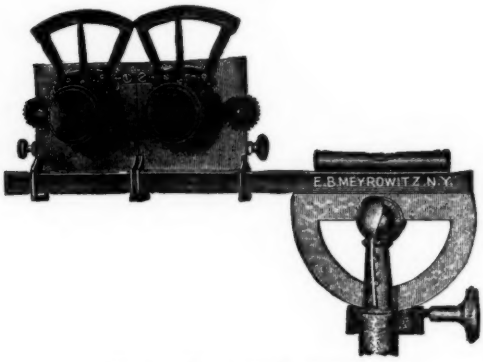

FIG. 7—The Lens Clinoscope.

cases either of heterophoria or of strabismus it will be easy to determine the kind and degree of declination and that the necessary correction will be as easy.

In heterophoria it is almost always easy to determine the form and extent of the manifest declination and hence the method of correction should that be determined on. It is not so easy to determine the declination in strabismus. Indeed in a certain percentage of cases no form of instrument which I have tried will give results that can be relied upon.

In the majority of strabismus cases in which we can obtain aid by means of the clinoscope, the lens clinoscope is by far the most useful instrument. The tube is so very short and the lens so strong that the patient will, in very many cases, give us information which is consistent and practically correct,

although no such information could be obtained by the clinoscope. Hence, although the lens clinoscope is of far less value than the clinoscope in ordinary cases of heterophoria, because of the fact that, even with the very small opening and of the short focus, there is a possibility of a leaning of an image caused by an oblique line of sight through a strong lens, a fault which can not be eliminated, yet in a case of strabismus after three or four examinations in which the results are practically uniform the result may be accepted until conditions have been so far improved that the clinoscope can be used.

In a certain proportion of cases, however, even the lens clinoscope is of no value, in some instances, because of the amblyopia of one eye; in others, and these are much more frequent, because the subject of strabismus can not be induced to get simultaneous vision in the two eyes. This is true even when the patient will declare that he sees the image before the right and the left eye at the same time. As a fact the adjustment of each eye is made not only independently of the other but at different periods of time. These different periods may approach so nearly to each other that the examined person will insist that they are simultaneous. In these cases we find an apparent absence of all declination. In fact were we to trust to the statements of the examined person we would find in many bad cases of strabismus an absence of declination which would be entirely exceptional even in case of the best adjusted eyes.

In such cases, if there is failure to discover the declination after a reasonable number of trials on different days, and if it is desirable to operate for the strabismus, there remains a measure to which one may tentatively resort even in cases of quite pronounced amblyopia.

The eyes being slightly cocainized, a very small square of white or black paper is placed upon the periphery of each cornea while an assistant holds the eyelids up in order that the small squares may not be swept away. A lighted candle being then placed in a position directly in front of the examined person at a distance at which the light can be seen by each eye, the examiner passes a small object, a visiting card or some such screen, alternately before each eye, and as each is uncovered the examiner observes with great care the movement of each

eye as it comes to fixation. Of course, in a case of convergent squint, the just uncovered eye will move outward and probably up or down. With these movements the observer need not concern himself. It is the rotation on the antero-posterior axis which should interest him and by very careful scrutiny of the movement of the little paper patch he will, if he has patience and skill in observing, be able to detect the wheel-like motion of the cornea and its direction.

Fortunately for such a method of examination the declination in a high degree of concomitant squint is *always very high*. Hence it is generally possible to obtain this decidedly rude yet extremely useful guide to the adjustment of the eyes.

There are still other cases in which the amblyopic eye does not make an attempt at fixation. In such cases we may tentatively assume that the eye with the negative declination is the inward squinting eye.

In the case of a proposed correction of the squint by operation, such a test as either of the above should, of course, be regarded as purely tentative. An operation with these findings in view might be undertaken only to the extent that a more satisfactory test might be made, when final measures depending absolutely upon definite knowledge may be adopted. In its place we shall return to a discussion of the examination and treatment in cases of squint, for while in most cases of moderate strabismus there is no great difficulty in determining the kind and degree of leanings of the meridians, in high degrees of squint, especially with amblyopia, the difficulties may be great.

If we inquire as to other effects of declinations, we shall find a broad field of inquiry with a great variety of resulting conditions depending upon many direct and collateral influences. The conditions of heterophoria and of heterotropia have already been mentioned, and since these conditions are themselves due to declinations it follows that the effects due to heterophoria are indirectly due to declinations. But many conditions are due to declinations directly which may be of slight or of weighty importance.

Among these conditions of apparently slight consequence we may find some which are in fact of very great moment.

Thus, an asymmetry of the face may appear to be simply a personal peculiarity. In fact this asymmetry may be so intimately associated with pains about the head as to be a matter of extreme consequence. Such associations have received practically no attention except as I have endeavored to emphasize their importance. Practically the question of habitual comfort or of customary distress may depend upon the expression of the face of the individual. The phrase that such or such a person "is so homely that it makes the face ache" has infinitely more of truth than the users of the phrase suspect.

An asymmetrical face is generally a face the sides of which are disproportioned by unconscious muscular efforts to overcome the visual confusion resulting from declination, and this habitual tension of certain facial muscles in the interest of correcting the leanings of the ocular meridians results in pain which becomes habitual with the individual who is forced to make the efforts.

Although I have referred to these facial tensions in various connections I am impelled to return to the subject which is of far greater importance than it would be supposed by one who has given it scant attention.

It is not practicable here to enter into the details of the results of declination on the facial expression to a considerable extent. One who would care to examine the subject further will find a more extended discussion in my work on *The Motor Apparatus of the Eyes* and in a series of articles published in the *New York Medical Journal of* 1910 and 1911. One or two phases of the subject may, however, be introduced here as illustrative of the general principle.

If one sits in one of our street or subway cars, so situated as to be able to study the faces of the occupants of the other side of the car, and if the observer takes critical notes of the peculiarities of these faces, it will very soon become apparent that a large majority of the faces are asymmetrical in the sense that one of the brows is more or less compressed, while the other is raised. This form of asymmetry is so general that, once carefully observed, it will rarely after that escape the notice of the observer.

Still more critical examination will impress the fact upon the

observer that a very large proportion of this asymmetry consists of a depression of the left and an elevation of the right brow (Fig. 8). Less frequently, other forms, to which we may refer, will be seen, but this is the prevailing type of facial expression so far as the appearance of the brows is concerned. With this peculiarity will often be observed other asymmetrical features, for example, the drawing of the mouth to one side, a greater fullness of one side of the face, and other peculiarities, generally less conspicuous than the one to which I have referred.

FIG. 8

Now, these asymmetrical features are, as a rule, if not invariably, the result of certain tensions of the facial muscles induced in the interest of maintaining a correction of declination. It is not a matter of surprise to one who has thought of this class of facts that a large proportion of those who show this asymmetry in a high degree are subjects of pains in the region of the brows. On this point we can here allude only to the fact. Later we may return to the subject. In another form of peculiarity of facial expression we see both brows raised toward the temporal side, while in still another class the temporal ends of the brows are depressed while the medial ends are elevated. (Fig. 9). With this last form there is nearly always the associated facial expression of deep vertical folds between the brows extending upward upon the forehead.

FIG. 9

All these and still other forms of facial expression are the direct results of declination and all are the result of more or less intense muscular tension in the effort to correct the defect. Elsewhere I have endeavored to indicate the especial muscular forces which are brought into action in the case of these peculiarities.[1]

We have space for only one inquiry here. Why, if these facial expressions are the result of declination, should one form of facial expression prevail to a markedly greater extent than another?

It is very easy to reply to this inquiry that clinical experience

[1] *N. Y. Med. Journal*, May 13 and 17, 1911.

shows that a combination of adjustments is prevalent in which there is little or no declination for the right eye, and that little when present of a negative character, while there is from a moderate to a high degree of positive declination for the left eye. It is safe to say that this statement is correct for more than four out of every five cases that may be examined whether the subjects of the examination are free from nervous reactions or not.

Whatever may be the reason for this fact it is beyond question an actual fact.

If we attempt an explanation I can resort only to an hypothesis.

It is known that as a rule the development of the left cerebral lobe is materially greater than the right; that its weight in adult life is considerably greater than its fellow. There are exceptions to this rule but it is nevertheless the rule.

It is more than probable that this greater development of one side of the brain is a result of the more active exercise of the muscles of the right side of the body than of the left side during the period of early growth of the individual. In other words, the excess in size and weight of the left side of the brain is due to the fact that the child is taught in very early life to be right-handed.

Now, with a more rapid development of the left brain in the plastic years of growth there occur a pushing outward of that side of the cranium and a tilting of the apex of the left orbit toward the temporal side, resulting, of course, in a tilting of the meridians of the left eye in the same direction.

Also, if the development of the left side is considerably more rapid than that of the right, then there may and often will be a tilting of the apex of the right orbit somewhat to the medial side, with a corresponding result to the globe of the eye.

Hence, though we have for both eyes a position of the meridians which is not as favorable to the functions of the various adjustments of these organs, we have not necessarily any disease, weakness, or disability of any kind of any muscle or muscles within the orbit.

That there are other cases in which both orbits tilt outward, and yet rare cases in which it is the right orbit that tilts to the temple, is true, but they are far less frequent than the first

mentioned condition. I am accustomed to inquire, of the persons who have the exceptional condition of positive declination for the right and negative or no declination for the left, if they are left-handed. To this I have not received uniform replies. In many cases the coincidence is established. In some it is not. I suspect that in these later cases the individual has had the habit of left-handedness broken at an early age, but not before the asymmetry of the orbits has been established.

If the hypothesis briefly indicated above is a well grounded one it would lead naturally to the conclusion that ambidexterity has marked advantages in this respect. From long-continued observations I am convinced that it has great advantages in many respects.

STUDIES ON THE ACTIONS OF TOXINS AND PROTEIN DEGENERATION PRODUCTS ON THE EYE.

II. The Action, Nature, and Source of the "Uveal Poison" of Ferment-Producing Bacteria

By Dr. ALAN C. WOODS, Philadelphia.

(From the John Herr Musser Department of Research Medicine, University of Pennsylvania.)

IN 1908, Stock (1), by the intravenous injection of yeast, produced a slight ocular inflammation characterized first, by a papular iritis, and, in the course of a few months, by a choroiditis disseminata. In 1911, Guillery, working in Cologne, influenced by this earlier work and no doubt also by the great attention then being given to ferments, began his work on the action of these bodies on ocular tissue.

By the injection of solutions of zymase, trypsin, and papayotin into the vitreous (2), he obtained inflammatory changes limited largely to round-cell infiltration of the choroid. Realizing that many bacteria were ferment producers, he prepared filtrates of cultures of the most promising, notably B. pyocyaneus and a staphylococcus. These filtrates were proved to be sterile by subculture and their ferment activity was tested by their power of liquefying gelatin. Intraocular injection of these filtrates gave inflammatory changes, limited again almost entirely to the uveal tract, the most notable feature being the round-cell infiltration.

Guillery was impressed by the pathological similarity of this picture to that of sympathetic ophthalmia and in further work, reported in 1912 (3), followed up this line of thought. He inoculated the vitreous of the excised horse's eye with ferment-

producing organisms, incubated this, filtered through porcelain, and tested the filtrate for sterility and ferment activity. Injection of this into a rabbit's eye gave the same results as before noted, but followed in this case by a sympathetic inflammation in the uninjected eye. He then attempted to determine whether or not this sympathetic inflammation was of anaphylactic origin. A rabbit was sensitized by intraocular injection, and after the proper period was reinjected in the blood stream. The results were negative. A second intravenous injection caused a lighting up of the process in the injected eye, while a third intravenous injection gave inflammatory symptoms in the uninjected eye. Next, using the filtrate of cultures of B. prodigiosus, grown on 15 per cent. gelatin containing 1 per cent. peptone, he obtained even more prompt and constant results.

He then tried intravenous injections alone of this filtrate of B. prodigiosus, grown on the gelatin medium. Frequently, in as short a time as forty-five minutes, a pericorneal injection appeared in the upper part of the orbit, with diminution in intraocular tension and signs of uveal irritation and inflammation. At this time he stated his conclusion that a picture very similar to sympathetic ophthalmia could be produced by the intravenous injection of this ferment poison.

In his third (4) and fourth papers (5), published in 1913 and 1914, he showed that this poison is common to the filtrates of practically all the ferment-producing bacteria. He claimed that the poison, while it had a certain general toxic action, was, in the eye, specific for the uveal tract. Practically all rabbits injected reacted similarly, although some few reacted only after the second or third injection. Rabbits receiving intravenously *2cc* of this toxin every third day, over a period of six weeks, gave the picture of a chronic iridocyclitis. The pericorneal injection started usually within a few hours following the intravenous injection and showed an exacerbation after each injection; occasionally slight exudates were observed in the anterior chamber—in short the symptoms of a chronic iritis, and generally a diminution of intraocular tension. On section these eyes showed a chronic infiltrative uveitis, with collections of round cells about the ciliary processes, lymphocytic infiltration of the choroid, thickened iris, and œdematous

ciliary body, with an increase in the connective-tissue elements. Frequently small hemorrhages were observed in the ciliary body. Blood cultures, taken during life and at autopsy, were negative.

In his latest paper in 1914 (6), Guillery retracts his former assertion concerning the relationship of this poison to sympathetic ophthalmia, but reiterates his opinion as to the toxicity on the uveal tract of this substance which he calls *Uveagifte*. Furthermore, by citing the opinion of Frank, prosector in the Augusta Hospital, he answers the criticism of Fuchs, that the round-cell infiltration, emphasized, could be observed in sections of the eyes of normal rabbits. Frank believes that the round-cell infiltration beyond doubt exceeds that found in normal eyes, and that it is clearly evidence of a chronic inflammation. Guillery makes no attempt to go into the nature of the poison, beyond stating that it is directly proportional to the ferment activity of the filtrates, which was measured by the power of liquefying gelatin.

EXPERIMENTAL RESULTS OF INTRAVENOUS INJECTION OF THE POISON.

With the idea of confirming, or otherwise, these observations of Guillery, and, if possible, of establishing the origin and nature of this poison, we have repeated, to a certain extent, his experiments. In the preliminary work, both B. pyocyaneus and B. prodigiosus were used, but as essentially similar results were obtained with both organisms, B. prodigiosus was finally selected as the organism to be used in preparing the various filtrates, autolyzates, and suspensions.

Throughout the experiments several difficulties were encountered, not emphasized by Guillery. Most of the solutions and suspensions prepared were extremely toxic to the animal. This general toxicity was in direct proportion to the ocular reaction—that is, the filtrates and suspensions giving the most marked ocular reaction were most poisonous to the animal. Many rabbits died shortly after injection, and in general these rabbits showed the most stormy ocular reactions. Conversely, those poisons which could be injected repeatedly with little or no general effect caused but slight ocular reactions.

In no case could rabbits be injected over a period longer than four weeks without causing death, and in these rabbits the ocular reactions were relatively mild. In this respect, this work differed from that of Guillery, who reports experiments in which rabbits were injected over periods of seven weeks, the rabbits showing rather stormy exacerbations of the ocular symptoms following each reinjection.

The general symptoms observed following injection were in most cases similar, irrespective of the poison used, varying only in intensity according to the degree of toxicity of the solution injected. Within a short time after injection, from a few minutes to an hour, the rabbit would appear uneasy and become slightly dyspnœic. A marked diarrhea frequently appeared within an hour. If the solution used was not especially toxic, the animal would gradually recover and appear normal within a few hours. In other cases, however, the diarrhea would persist and the rabbit would become prostrated. Sometimes the rabbits survived this stage and recovered, but generally the respiration became more labored, and death followed after a period of from a few hours to several days. Blood cultures taken during life and at autopsy were constantly negative. Albumin was occasionally found in the urine.

Autopsy showed always an extreme vaso-dilatation, splanchnic engorgement, hypostatic congestion of the lungs, congestion of the liver, kidneys, and spleen. Frequently large hemorrhages were found in the colon and small intestines, less frequently in the adrenals, mesenteric lymph nodes, and pancreas, and small petechial hemorrhages throughout the cortex of the kidney. With one exception the hemorrhages were noted only as the result of administering those poisons in which the enzymes had not been killed by heating. Histologic study of the tissues showed this same engorgement and hemorrhages, and, in cases when the injections had been long continued, a slight tubular nephritis.

The ocular reactions obtained confirm in the main the results reported by Guillery. Thus, following Guillery's technic (intravenous injections of *2cc* every third day), ninety per cent. of the animals injected show ocular reactions, usually within a short time after injection. Generally within from thirty

minutes to two hours after intravenous introduction of the poison, a pericorneal injection appears around the upper part of the cornea, in the region of the insertion of the superior rectus muscle. The vessels of the iris become extremely dilated and the reaction of the pupil sluggish. In cases in which the solution injected was especially toxic, a general pericorneal injection of the fine capillaries surrounding the cornea is observed, the iris becoming thickened and losing all luster. Occasionally a slight clouding of the cornea is observed. In other cases the primary pericorneal and iris injection persist for a period of from a few hours to a few days without further symptoms. On reinjection, these animals usually show an excerbation of these same symptoms, more or less intense than the primary reaction. A few animals seem to develop gradually an immunity to the toxin; at least, after several injections they fail entirely to react.

Histological study of these eyes show, in the main, the lesions reported by Guillery. The most constant finding is a very definite collection of lymphocytes around the ciliary processes. This is clearly far beyond that observed in a large number of normal rabbits' eyes examined. Less frequently is seen the round-cell infiltration of the ciliary body and choroid, emphasized by Guillery. In a number of the eyes, small hemorrhagic areas are scattered throughout the ciliary body and processes. Slight fibrinous exudates are in some cases found around the ciliary processes and on the posterior surface of the iris, at times forming what are apparently posterior fibrinous synechiæ. In no case are exudates found in the anterior chamber. In some cases, the iris appears œdematous with greatly dilated vessels. In many cases a proliferation of young connective tissue occurs.

The results obtained in all experiments with various preparations of the toxin were practically the same, varying only in intensity. The following protocol is that of a typical experiment.

Toxin. B. prodigiosus was grown for seven days on peptone free broth, 0.5 per cent. acid. The culture was then filtered through a Berkfeld filter and proved sterile on subculture.

Rabbit Z—14. February 16th. Eyes appeared normal

to external and ophthalmoscopic examination. Intravenous injection of *2cc* of the toxin in the marginal ear vein, at 11.00 A.M. *11.30* A.M. Eyes negative, general condition good. *1* P.M. Moderate pericorneal injection of both eyes, rabbit slightly dyspnœic. *4* P.M. Marked diarrhea. Moderate pericorneal injection of both eyes and dilatation of vessels of iris. Reaction to light sluggish. Slight clouding of cornea of right eye, and definite photophobia.

February 17th. There is still a moderate pericorneal injection. The process is fading somewhat.

February 19th. The eyes appear normal, with exception of a slight pericorneal injection. Intravenous injection of *2cc* of toxin at 4 P.M. *5* P.M. General condition fair. Eyes show no further reaction. *5.30* P.M. Animal is dyspnœic and has a marked diarrhea. Eyes show a moderate injection of fine capillaries around cornea.

February 20th. Appears in good condition. Eyes show only slight pericorneal injection. Iris, cornea, and fundus negative.

February 22d. Eyes are unchanged. Intravenous injection of *2cc* toxin at 1.30 P.M. *2* P.M. The rabbit is slightly dyspnœic—there is a moderate pericorneal injection.

February 23d. There is a moderate pericorneal injection, and slight photophobia; general condition appears good.

February 25th. The eyes show only a slight pericorneal injection. Intravenous injection of *2cc* toxin at 12.30 P.M. *1.30* P.M. The pericorneal injection is more marked, and there is slight photophobia. General condition only fair. *4* P.M. Condition unchanged.

February 28th. Eyes show a slight pericorneal injection. Intravenous injection of *2cc* toxin at 12 M. *5* P.M. The rabbit is dyspnœic and has a diarrhea. Eyes show a moderate pericorneal injection and photophobia.

February 29th. *9* A.M. Rabbit is very ill, lying on side. Left eye shows moderate pericorneal injection. Right eye shows a lusterless, thickened, and inactive iris, marked pericorneal injection, and slightly steamy cornea. *12* M. The rabbit continues very prostrated. Both eyes show marked pericorneal injection, thickened, inactive, and lusterless iris, and slightly steamy cornea. *2.30* P.M. Condition is unchanged. *5* P.M. Rabbit is very weak; eyes show no further change.

March 1st. The rabbit is in the same prostrated condition; no change in eyes. Died during the night.

Autopsy shows a marked splanchnic engorgement, with congestion of the liver and spleen. The cut surface of the kidney is pale and lusterless, with apparently some cloudy swelling. The adrenals are large and soft, and have min-

ute hemorrhages over the surface. Autopsy is otherwise negative.

THE NATURE AND SOURCE OF THE POISON

Guillery showed that the ability to produce this poison is common to all ferment-producing organisms, and that the degree of toxicity is proportional to the ferment activity of the prepared poison, and assumed that the toxic action of his poison is due to the ferment itself.

In order to ascertain primarily whether the toxicity of this poison was due to ferments or to other toxic substances we have taken advantage of the fact that enzymes, or ferments, can stand a temperature of 100° or more when dry, but when in solution are generally destroyed somewhat below 70° (Wells). The filtrate of B. prodigiosus, grown on a gelatin medium, was prepared as described, and was then heated at 65°–70° for two hours. At the end of this time it had lost the power to liquefy gelatin. Intravenous injections of 2*cc*, however, called forth exactly the same general and ocular symptoms as did the fresh filtrate. In later work therefore all filtrates, suspensions, and autolyzates were divided into two portions, and the ferments in one portion killed by heating. On the injection of these, no difference was observed in the local or general action of the two solutions, except that in general, probably slightly more intense ocular reactions foll wed the injection of the heated portion.

It is evident, therefore, that the prepared solutions contained a definite poison entirely independent of ferment content. The fact that this poison is produced in common by ferment-producing organisms renders it highly probable, however, that its formation is dependent upon, in some way, the activity of ferments.

The various possibilities, therefore, are:

I. That the poison is inherent in the culture medium itself.

II. That it is formed through the transformation of some constituent of the medium, through ferment or some other activity.

III. That it is the result of the metabolism of the organisms themselves. In this case it would be either (*a*) a true

toxin secreted in the medium, or (*b*) a degeneration product of the bacterial body, freed into the medium by autolysis.

I In order to determine the first of these points, the gelatin solution used by Guillery (fifteen parts gelatin, eighty-five parts of 1 per cent. peptone meat infusion broth) was injected intravenously in rabbits in 2*cc* doses every third day, over a period of eight weeks. No reaction was observed. Similarly ordinary 1 per cent. peptone meat infusion broth was injected intravenously in a like manner. No reactions were here observed The rabbits showed no ocular or general symptoms, remained healthy, and gained weight.

II. To determine whether the ocular and general reactions observed were due to secondary degeneration products formed in the medium or in some component part, the medium was gradually simplified, the filtrates being prepared as usual. First gelatin and then peptone were omitted. The organisms were finally grown on a meat infusion peptone-free broth, containing 5 per cent. NaCl. On this simple medium, B. prodigiosus grew well. The filtrates of culture grown on these various media all gave the same toxic effect, although in general the reactions were less intense as the medium was simplified.

The organism was finally grown on Uschinsky's culture medium, which contains no proteins, carbohydrates, or fats.[1] B. prodigiosus grew very poorly on this medium, and after five days but a light growth was present. It was then filtered through porcelain and proved sterile on subculture. Intravenous injection of this filtrate in 2, 4, and 6*cc* doses gave no appreciable ocular or general effect. Since bacteria such as the diphtheria and tetanus bacilli, which secrete true toxins, produce their toxin regardless of the medium in which grown, the poison in question evidently does not belong in this class; though the growth on Uschinsky's medium was so light that the production of any toxin must necessarily have been small, and for this reason this experiment does not seem entirely conclusive. However, in view of the lack of toxicity of this filtrate

[1] Uschinsky's culture medium is: Water 1000 parts; glycerin 30–40; sodium chloride, 5–7; calcium chloride, 0.1; magnesium sulphate, 0.2–0.4; di-potassium phosphate 0.2–0.25; ammonium lactate, 6–7; sodium asparaginate 3–4 parts.

combined with the proven toxicity of filtrates from cultures grown on peptone-free broth, and, in view of the decreasing toxicity of the filtrates, as the medium on which the organism was gradually simplified, it is safe to conclude that the poison is formed, at least to a certain extent, through the transformation of the protein constituents of the culture medium by the proteolytic activity of the bacterial ferments.

III. To determine whether the poison is due to the products of the bacterium itself, B. prodigiosus was grown on agar slants in 100*cc* test tubes for one week. The growth on each tube was then washed off with 10*cc* of sterile salt solution. This suspension of B. prodigiosus was divided into two portions. One portion was heated to 65°–70° for two hours, killing all organisms and ferments, and was proved sterile on subculture. The other portion was unheated. Intravenous injection of 2*cc* of each of these suspensions called forth ocular and general symptoms. Suspensions of organisms grown for three days on agar slants, and similarly treated, gave even more severe reactions than the organisms grown for one week. Suspensions of organisms grown for twenty-four hours gave only very moderate reactions.

It is evident, therefore, that one certain source of the poison is the bacteria themselves. Though the bacterial body is one source of the poison, the poison is also developed by the bacteria in the medium in which they are grown, and is therefore manifestly not an endotoxin. Endotoxins do not escape from the cells into the culture medium, but are held firmly within the cells (Wells). The poison must theoretically be, therefore, either a soluble toxin or a soluble degeneration product formed from the protein of the bacterial body, or from the protein of the medium.

The failure to produce symptoms by the intravenous injection of the filtrate of cultures grown on Uschinsky's medium make it highly improbable that anything in the nature of a true toxin is excreted. Yet, as before noted, the growth on Uschinsky's medium was so slight that the results must be somewhat discounted.

To exclude absolutely, therefore, the possibility of the poison being a true soluble toxin, not destroyed by heat, the organisms were grown for two days on agar slants, washed

off with salt solution, and the resulting suspension immediately heated to 65°–70° for two hours to kill organisms and ferments. This suspension was then incubated at 37° for twenty-four hours. At the end of this time the organisms were removed by centrifugalization, and the supernatant fluid filtered through porcelain to remove any remaining organisms. The resulting filtrate was sterile on subculture. It contained only such soluble materials as could be dissolved from the bacteria by a process of simple solution. Intravenous injection in rabbits gave uniformly negative results with one exception. This exception was a small rabbit, 1020*grms.* in weight, which received an intravenous dose of 5*cc.* In two hours it showed slight uneasiness and diarrhea, and the slightest possible pericorneal injection, all of which symptoms rapidly disappeared, and in four hours the animal appeared wholly normal. In explanation of this, it is probable that, during the period the organisms were grown on agar, certain degeneration products may have been formed, which were taken into solution during the time the suspension was incubated (or it is possible that not quite all autolytic ferments were killed during incubation); either of which would explain the slight reaction following a large dose in a small rabbit. It was clear, however, that the poison under investigation was not a true soluble toxin and was not formed to any extent independently of proteolytic or ferment action.

Further, to prove the probability of the poison being a degeneration product formed by the action on the bacterial body of a proteolytic ferment, B. prodigiosus was grown two days on agar slants, and the organisms then washed off with sterile salt solution. This suspension was at once incubated, without heating and destruction of the ferments, and autolyzed for forty-eight hours. At the end of that time the organisms were removed by centrifugalization, and the supernatant fluid filtered through a porcelain filter, and proved sterile on subculture. This clear filtrate was divided into two parts, and one portion heated two hours at 65°–70° to destroy autolytic ferments. The other portion was untreated. Intravenous injection of 2*cc* of both these solutions called forth prompt and stormy ocular and general symptoms, identical with those described. One 1500*grm* rabbit died within four

hours after injection, with diarrhea, dyspnœa, and prostration, showing within forty minutes after injection pericorneal injec ion, lusterless, œdematous, and congested iris, lacrimation, and marked photophobia.

To exclude the possibility of this reaction being due to some product removed from the medium with the bacterial bodies, the organisms were washed off a three-day agar culture in the usual manner. They were then thrown down by high speed centrifugalization, the supernatant salt solution pipetted off, and the organisms again stirred up with fresh salt solution. This was repeated three times in order to thoroughly wash the organisms. They were then taken up in 10*cc* salt solution, and autolyzed for forty-eight hours. The organisms were then removed by centrifugalization, and the supernatant fluid filtered through porcelain, and proved sterile on subculture. Intravenous injection of this gave the usual symptoms with an intense ocular reaction. These last exp riments proved conclusively, that the poison can be developed from the bacterial bodies, that the production of the poison is dependent upon the action of the bacterial ferments during autolysis and not to any inherent substance taken from the culture medium.

DISCUSSION.

The poison is evidently con ained, in as high a degree of concentration obtained in any solu ion, in the clear sterile autolyzate of living B. prodigiosus. The poison is quite clearly a result of autolysis of the bacterial cell by the bacterial ferments. It is quite probable that it is formed also by similar autolytic action on the protein of the media on which the bacteria are grown. The positive results obtained with the filtrates of cultures grown on peptone-free broth, and the negative results obtained with the filtrates of cultures on Uschinsky's media, make this the more probable.

The poison probably comes under the class of ptomaines, protein degeneration products formed by the proteolytic action on media and on bacterial protein itself. These are soluble nitrogenous substances, commonly found in decomposing proteins. They contain nitrogen radicals, principally amino groups, and are closely related to the amino acids.

They are themselves subject to decomposition and on prolonged autolysis and in old cultures gradually disappear.

In this last respect this poison behaves similarly. It was present in only a slight degree in young cultures, reached its maximum development in three to four days, was present to less degree in ten days, and was not found in filtrates of cultures three weeks or more old. Moreover, the production of ptomaines is in no way parallel with the pathogenicity of the organisms producing them, highly poisonous ptomaines being produced by non-pathogenic organisms—a fact of interest in that B. prodigiosus is notoriously harmless.

The probability that this poison results from the decomposition of protein, and that circulating in the blood stream it can cause symptoms of chronic uveitis, offers attractive possibilities for proof of the theory of endogenous iritis resulting from gastro-intestinal auto-intoxication. The point remaining to be proved is the possibility of absorption of this toxin from the gastro-intestinal tract, and from serous cavities. Further experimental work along these lines is now in progress.

The ocular symptoms resulting from the intravenous injection of this poison are what should be expected following a single insult in a normal animal. The poison has a definite irritant action upon the uveal tract, and upon intravenous injection brings out symptoms of ciliary irritation. These symptoms persist at their maximum while the poison is still circulating in the blood stream. Upon excretion of the poison the cause of ciliary irritation is removed and the eye gradually returns to a nearly normal condition. On reinjection the eye again reacts to the fresh insult. When the toxicity of the poison is great, the uveal irritation is proportionately greater, and the eye shows a correspondingly more intense reaction, which persists until the death of the animal.

The large hemorrhages observed in the intestines, in the lymph nodes and in other organs, especially in the rabbits injected with solutions in which the ferments had not been killed by heating, are similar to those observed by Hildebrandt (7) and others, who studied the action of various enzymes, and are probable due to the actions of the ferments themselves.

No exhaustive studies of the physiological action of the

poison have been made, but the most significant finding in the dog has been a drop in blood pressure of 15 to 20*mm* on intravenous injection of the poison.

SUMMARY.

Poisons can be isolated from non-pathogenic ferment-producing bacteria, which have a definite action upon the uveal tract in the eye. The action is that of an irritant; and gives rise to an exacerbation of symptoms on each reinjection. The ocular symptoms consist of a pericorneal injection coming on within forty minutes to two hours after intravenous injection, followed, when the toxicity of the solution is great, by an injection of the vessels of the iris, sluggish or inactive iris, slightly irregular pupil, and occasionally slight clouding of the cornea. Histologically, the eyes show a round-cell infiltration about the ciliary processes and frequently a round-cell infiltration of the ciliary body and choroid, small hemorrhages in the ciliary body, and a slightly œdematous iris, with occasional posterior fibrinous synechiæ.

The poison produces general symptoms of uneasiness, slight dyspnœa and shivering, marked diarrhea, and, where the toxicity of the poison is greatest, prostration and death. The most evident lesion is marked venous congestion, and frequently large hemorrhages throughout the body. The hemorrhages were most frequently observed when the animal was injected with solutions in which the ferments had not been killed by heating. In the dog, the poison gives a decided drop in blood pressure. The chief source of the poison is probably the body of the bacteria itself, from which it is formed by proteolytic action of the bacterial ferments. It is also probably formed to a certain extent from the protein of the culture media itself. It reaches its maximum development in three to four days, and then gradually disappears, being entirely absent in cultures three weeks old. The poison seems to be a degeneration product of protein. It conforms to the class of ptomaines, basic nitrogenous compounds found in decomposing proteins. The action and origin of this poison offer distinct possibilities for experimental confirmation of the theory of auto-intoxication in endogenous uveitis.

CONCLUSIONS.

I. A uveal poison can be obtained from cultures of B. prodigiosus, and probably from similar ferment-producing organisms.

II. This poison is not due directly to ferment activity, but to a degeneration product formed from the bacterial body and the protein of the culture media by the proteolytic action of the bacterial ferments. The poison comes under the class of ptomaines.

III. Intravenous injection of this poison gives clinical symptoms and histological lesions of a chronic infiltrative uveitis.

IV. The poison is generally toxic to the animal, producing uneasiness, slight dyspnœa, diarrhea, and prostration.

V. The ocular reactions observed following the intravenous injection of the poison are directly proportional to the general symptoms produced in the animal.

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ILLUSTRATING DR. HECKEL'S ARTICLE ON "A CASE OF MELANOSARCOMA OF THE ORBIT, TREATED WITH RADIUM"

FIG. 3.

FIG. 2.

FIG. 1.

REPORT OF A CASE OF MELANOSARCOMA OF THE ORBIT, TREATED WITH RADIUM.[1]

By EDWARD B. HECKEL, M.D., Pittsburgh, Pa.

(With three photographs on Text-Plate XXIV.)

(From the Service of the Allegheny General Hospital.)

The subject of the following observation was seen first on April 16, 1912. The patient a man forty-eight years of age. There was no history of any eye injury; general good health had always prevailed. The right eye had never annoyed him; the left eye, however, was and had been blind for twelve years; the condition then present had commenced thirteen years before: "it simply got weak."

Inspection showed the right eye negative in every respect with a corrected vision of $\frac{20}{20}$, w. + 0.75 D. C. ax. 180°. The left eye: the cornea flat, yellowish, and opaque with some blood-vessels in its substance. It appeared like an eyeball which had undergone a sloughing of the cornea following a purulent conjunctivitis, although the patient denied any inflammatory condition of the eyeball at any time. The eyeball was somewhat shrunken and soft. An immediate enucleation was advised, but was refused by the patient, as he had heard "the removal of one eye would injure the other."

The patient returned, however, on September 9, 1913, for an enucleation of the left eyeball, because it had become painful. The right eye had remained as when first seen in April, 1912. The enucleation was done September 10, 1913. Quite a large dark staphylomatous mass was found protruding down and backward from the region of the equator of the eyeball. The eyeball with the projecting mass was carefully removed and the specimen sent to the pathological

[1] Read at the meeting of the American Ophthalmological Society, Washington, D. C., May 9, 1916.

laboratory, where it was examined by Dr. Haythorn, the pathologist, and pronounced a melanosarcoma.

The orbit healed promptly and the patient told of the diagnosis and probable prognosis and that very likely there would be a return of the neoplasm. As the patient was very desirous of wearing a prosthesis, permission was granted with a full explanation that the presence of the foreign body might act as a source of irritation. He was also cautioned that if at any time any pain or discomfort developed, or if the orbital contents seemed to increase, or if the prosthesis should appear to bulge forward, to report at once, as this would in all probability mean a return of the neoplasm.

He wore the prosthesis with comfort and satisfaction until March, 1914, or about six months after the enucleation, when he returned. At this time he presented the appearance shown in Fig. 1. The orbital contents had decidedly increased. The prosthesis was pushed forward and downward with a distinct bulging of the upper and lower lids. Palpation revealed the orbit to be quite well filled with a spongy mass limited only by the bony walls of the orbit.

Previous experience with orbital exenteration for malignant disease of the orbit decided the writer to try radium in this case. Consequently on March 27, 1914, a deep incision was made into the orbit, practically bisecting it in its horizontal meridian, and fifty-three milligrams of radium element in a silver capsule, screened by *2mm* of lead and covered with a rubber tube, inserted into the orbit in its anteroposterior diameter and held in place by means of a light compress and bandage and left in place for eleven and one half hours.

The presence of the radium with its screening was somewhat painful but did not cause any great discomfort. There was no febrile reaction. On April 13th, or seventeen days after the exposure to the radium rays, a distinct mass could be palpated in the center of the orbit. This mass was dissected out on the following day and proved to be strongly pigmented and $3 \times 2\frac{1}{2} \times 2cm$ in size. This was sent to the laboratory and the same diagnosis was made as was made of the enucleated eye, melanosarcoma. The orbit was lightly packed and two days later fifty-three milligrams of radium element, screened as before, was inserted and allowed to remain in place for thirteen and one half hours. The orbit did nicely after this with a slight sloughing discharge. The orbit was kept clean and healed promptly. It showed no evidence of any return of the neoplasm, at least there was no increase in the orbital contents, and after a few months presented the appearance shown in Fig. 2.

In November, 1914, the orbit became somewhat painful,

especially at night, and there was a slight increase in its contents, especially along the nasal side. Palpation revealed a soft mass.

On December 7, 1914, quite a bit of strongly pigmented material was removed by curetting, and four days later fifty-three milligrams of radium element, screened as before, was inserted and left in place for twelve hours. This as before was followed by some sloughing from the orbit. The patient did nicely for six months or until June, 1915, when the orbit again became painful and palpation revealed a movable mass within the orbit. On June 11, 1915, the orbit was again curetted and one hundred milligrams of radium element, screened as before, was inserted and left in place for fourteen hours. This, as before, was followed by a sloughing from the orbit. The orbit continued to do nicely and when the patient was seen on July 12, 1915, he complained of nothing except a burning sensation which he had not observed before.

The patient was not seen again until December 22, 1915, when the socket was partly filled, clean, and quiet and the surrounding structures quite normal, as shown in Fig. 3. This photograph is slightly misleading as in reality there was quite a depression while the photograph makes the orbit appear quite full. The patient, however, appears pale and anæmic. He stated that he had lost 25 lbs. in weight but felt quite well. There was no pain or discomfort about the orbit. His blood was examined at this time by Dr. Haythorn with the following result:

Red cells, 4,716,000; leucocytes, 6360. *Hg.* 80%. Differential; neutrophiles, 63.5%; eosinophiles, 4.5%; basophiles, 1%. Mononuclear: small 9%; large, 12.5%. Transitionals, 1.5%. Unclassified, 8%.

The patient was seen last on April 25, 1916. The orbit has remained quiet, no pain or discomfort and no increase in its contents. His general health, however, has been rather poor: he has lost some weight and was confined to his bed during the entire month of February. He now weighs 114 lbs. his normal weight being 150 lbs. His physician, Dr. Midgley, under date of May 5, 1915, tells me "that there is very extensive involvement of the liver, spleen, and pleura and probably of the bowels."

The value of radium, if used in sufficient amount, in malignant disease of the orbit is, in the opinion of the writer, clearly demonstrated by this case. The radium kept this neoplasm of the orbit in absolute abeyance, and the patient in a condition to follow his vocation up to within the last three months

or during a period of two and one third years. The experience of this case has also suggested to the writer the use of the electric-cautery knife, at a cherry-red heat so as not to carbonize the tissues, for the removal of the malignant mass from the orbit instead of the knife, as has been suggested by others for the removal of malignant tissue from other parts of the body, so that when a similar case presents itself the writer will pursue the following technic: Make an incision through the skin of the lids about 5*mm* from the free border of lids, completely encircling the palpebral fissure, then dissect the skin only of the lids back to the bony orbital wall, then with an electric-cautery knife, at a cherry-red heat, by repeated punctures directed toward the apex of the orbit and as closely to the bony wall as possible, completely sever the cone-shaped orbital content, and then introduce one hundred milligrams of radium element and allow it to remain in place twelve hours, the radium to be introduced again at some future time if necessary or advisable. The writer is strongly convinced that one application of one hundred milligrams of radium element is of more value than several applications of a less amount.

The writer is indebted and hereby expresses his appreciation and gratitude to Dr. Wm. H. Cameron of Pittsburgh for the generous loan of the radium used in this case.

RETROBULBAR NEURITIS WITH CENTRAL SCOTOMA FROM TOXIC ACTION OF THYROIDIN.[1]

By Dr. MYLES STANDISH of Boston.

THYROIDIN and thyroid extracts have attained so large a place in our armamentarium that, if they are capable of producing optic neuritis involving the macula bundle of fibers, it seems desirable that this fact should be well established.

The number of cases reported to date, considering the increasing use of the drug in the last few years, has been very small. It is possible that, with other conditions present, the cause of the neuritis has been overlooked.

I have been able to find but eight cases of retrobulbar neuritis with central scotoma which have been attributed to the use of thyroid extracts.

Dr. Henri Coppez in *Arch. d'ophtal.*, 1900, reports five cases. There were four women and a man between thirty and forty years of age. They developed the toxæmia after using the drug in large doses for some months. They had all lost a large amount in weight, but the only other symptoms were general nervous disturbances. There was in each case a loss of vision in both eyes to $\frac{1}{10}$ or less, but not an equal amount in each eye.

Dr. Coppez reports one case in full: A man thirty-six years of age had been taking thyroid tablets in large doses and had lost in eight months 26 kilograms in weight. The latter part of this time he had taken nine tablets daily.

The general conditions were nervousness, sleeplessness, and muscular weakness.

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[1] Read at meeting of the American Ophthalmological Society, Washington, D. C., 1916.

Ophthalmoscopically he had hyperæmic nerves without inflammation; there was venous stasis in the retinal vessels and slight pain from pressure on the globe. Vision in the right eye was $\frac{1}{12}$ and in the left eye $\frac{1}{6}$. Absolute central scotoma of about 10° in the right eye and a relative scotoma in the left eye. The periphery of the field was intact. He was forbidden thyroid, alcohol, and tobacco. After three months, vision in the right eye was ¼ and in the left ½, and he had in the right eye a slight central scotoma for red and green. The periphery of the field for white and color remained normal. After three months, vision in the right eye was ⅓ and in the left eye ⅔. The body weight had increased. The retinal veins were still slightly turgescent. It is unfortunate that this was the case of the man and that alcohol and tobacco were forbidden coincidentally with thyroidin. Coppez says that the disks were somewhat more altered in appearance than in alcohol and tobacco neuritis. The veins were often swollen and the arteries diminished in size.

In *Klin. Monatsblatter für Augenheilkunde*, 1911, Carl R. Hennicke reports a case of a man fifty-three years of age. First seen in 1907. He had a central vision of $\frac{6}{9}$ in each eye with a myopia of 0.50 diopters. The ophthalmoscope showed the left eye with normal fundus. The right disk was somewhat reddened. The outline of the nerve was not lost. The arteries were very small, veins of normal size. It was a case of myxœdema and had been under treatment five years. In June, 1910, vision in the right eye was $\frac{6}{30}$. Ophthalmoscopically as in 1907. The patient had taken thyroidin since 1903. He did not return till the 21st of September. Vision in the right eye was then movements of the hand and in the left eye was $\frac{6}{24}$. The right nerve was white, the arteries scarcely visible. The left disk was swollen and reddened and the outline blurred. The neighborhood of the nerve not clear, the arteries small and the veins swollen. Iodide of potash was prescribed and thyroidin was omitted and on November 21st, vision in the right eye was $\frac{6}{30}$ and in the left $\frac{6}{18}$. Hennicke thought the ophthalmic changes were not due to myxœdema, but to thyroidin. On January 27, 1911, vision in the right eye was $\frac{6}{30}$ and in the left $\frac{6}{12}$.

Dr. Emanuel Snydacher, in a clinical note in the *Journal of*

the American Medical Association (Oct. 14, 1911, page 1290), reports a case of a woman forty-two years of age who came in complaining that in bright light she had difficulty in seeing, all objects appearing more or less hazy; but in a subdued light, or even in comparative darkness, she had no trouble.

An ophthalmoscopic examination showed an apparently normal fundus, with the possible exception of slight blurring of the edges of the disk. The vision was $\frac{2}{3}$ of normal in each eye.

Perimetric examination disclosed an oval central scotoma for red and green in both eyes. Investigation of her mode of life revealed no apparent cause for this condition. She denied being addicted to either tobacco or alcohol. Repeated examinations disclosed no sugar in the urine.

Quite accidentally during a subsequent examination, she volunteered the information that for a number of months she had been taking an anti-fat remedy recommended by a friend. Investigation revealed the fact that this patient had been taking thyroid extract for almost three months to reduce her weight. The thyroid extract was at once stopped; the patient was given strychnin hypodermically, and in a few weeks the scotoma disappeared.

Aalbertsberg reports a case of optic neuritis following the use of thyroid gland (*Ned. Tijdschr. v. Geneesk.*, ii., No. 22), a patient with myxœdema who had been treated with thyroid gland.

The effect on the disease was favorable, but the patient began to complain of poor vision in the left eye. Examination revealed optic neuritis. Later vision failed in the right eye and a low degree of neuritis was found here. The use of thyroid was stopped immediately and the condition of the eye improved. In the left eye, however, an atrophy of the nerve came on.

On May 7, 1910, a woman sixty-nine years of age came to see me; she had discovered a short time before that vision in the left eye was not as good as in the right.

She had a small central scotoma in the left eye and a central color scotoma in the right eye for green. Her vision was $\frac{6}{80}$ in the right eye and $\frac{6}{24}$ in the left eye. About ten days before she had first been unable to read newspapers.

With the ophthalmoscope there was found some slight swelling of the optic disk in the right eye, some narrowing of the arteries, but not marked turgescence of the retinal veins. There were several small retinal hemorrhages in each eye. It was originally a case of myxœdema, and I was told that formerly she had the features and club fingers which are markedly characteristic of the disease. When she came to me these characteristics had disappeared. She was anæmic, emaciated, weak, nervous, and looked like a very ill woman. She had been treated with thyroidin, and after several years' treatment the recovery was considered complete. However, she continued the drug on her own responsibility after ceasing to visit the doctor. For fourteen years she had taken 5*gr* thyroidin tablets three times a day, and for five or six years had taken one tablet each day.

Examination failed to find any disorder of the kidneys, or diabetes, and the physician in charge was inclined to attribute the retinal hemorrhages to her marked anæmia. Thyroidin treatment was discontinued and vision improved. I did not see her again, but was told the improvement was sufficient to enable her to thread a coarse needle, but there was a recurrence of the myxœdemic symptoms.

She died suddenly on the 19th of July.

On September 11, 1911, a man forty-one years of age was referred to me who had noticed in the previous January a blur before his right eye, and at the same time thought the vision in his left eye was not as good as formerly, and vision had continued to decrease. It was then $\frac{1}{10}$ in the right eye and $\frac{8}{10}$ in the left eye.

Six years before he had had one-half of the thyroid gland removed on account of a fibrous tumor, and four years afterward was supposed to have had the other half removed for the same cause. After the last operation he had a pulse of 120; after the first operation, there had been no increase of pulse. The outer limits of the field in the right eye were normal. He had a central color scotoma in the right eye for all colors except dark blue. He was slightly more excitable than formerly, and had some uncertainty in walking. There was no evidence of kidney disease or diabetes and no history of syphilis. No abnormal pupil symptoms.

He had taken since his last operation ½*gr* tablet of thyroidin before each meal.

Ophthalmoscopically the nerve was normal in color in each eye and vessels not markedly changed. He was advised to omit thyroidin and his vision returned in the course of a few months to normal in each eye. There has been a

return of the tumor of the thyroid, but his general condition is excellent.

On December 23, 1913, a woman who had been under my care eleven years for other ocular conditions, came to my office complaining of sudden loss of vision. She was thirty-three years of age, stout, and on advice of a friend had attempted to reduce her weight by taking a proprietary medicine. The composite name implied that it contained thyroidin. The medicine was put up in the form of a bolus which weighed 10*grs*. The only analysis of the medicine that I was able to find would seem to show that the bulk of the mass was probably desiccated thyroid. The label on the parcel said that the medicine "was designed for the treatment and reduction of excessive and abnormal fatty accumulations on the human body, and for the reduction of goiter and granular enlargements."

She had begun taking this five days before; had taken three doses on the 18th—three on the 19th—and six on the 20th. The following morning her vision was so reduced that she could not read and had remained so since.

Ophthalmoscopically both nerves were pale and the arteries small. There was a central scotoma in the right eye for all colors except a somewhat uncertain perception for blue. In the left eye the central scotoma was complete for all colors. There was no headache, nor nausea. Vision of both eyes was $\frac{20}{200}$. The pulse was 120. The nostrum was omitted, and on December 26th the pulse was 100, full and easily compressible. On January 26th, which was the last time I saw her, vision in the right eye was $\frac{20}{40}$ and in the left eye $\frac{20}{70}$ and the pulse 90.

Ophthalmoscopically both disks were somewhat whiter than normal, especially the left.

Birch-Hirschfeld and Inouye, *Graefe's Arch.*, lxi., p. 3, stated that experimental investigation on dogs shows the anatomical changes to be a degeneration of the ganglion cells of the retina with secondary degeneration of the optic nerve fibers.

It seems probable that in these three cases the retrobulbar neuritis was due to the thyroidin, as in each case the discontinuance of the drug was followed by a recovery of vision.

The most startling thing in this short series of cases is the wide difference in the toleration of the drug. One patient had taken the thyroidin in large doses for many years, and one patient had taken an unknown amount for only three days.

That the susceptibility to toxæmias varies greatly in different individuals and indeed in the same individual at different times is well known. In the case of tobacco a long series of years' use without harm may precede the toxic neuritis. The rapid production of the toxæmia in the last case is occasionally paralleled in other drugs.

I have hesitated about reporting the last case owing to the fact that one does not expect pathological changes of this sort to be due to an acute poisoning from drugs, but where so little is known of the toxic action on the optic nerve, it seems advisable that all cases in which the condition is suspected should be placed upon record. My principal object in reporting these cases is to place them on record, and to sound a word of warning that thyroidin should not be used indiscriminately for reduction of weight, and that the selling of proprietary medicines containing it to the public for this purpose is very likely a dangerous practice.

ILLUSTRATING DR. KNAPP'S ARTICLE ON "THE REMOVAL OF A TUMOR AT THE APEX OF THE ORBIT."

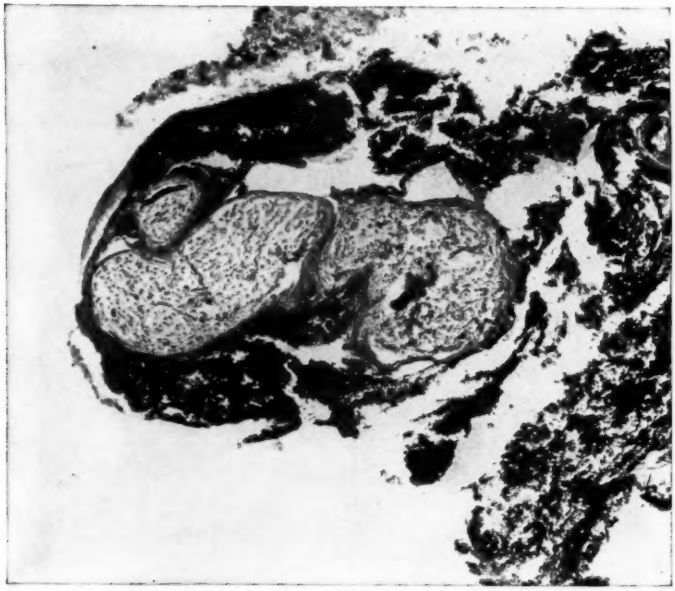

FIG. 1.—The Tumor (*Plexiform Neuroma*) of the Eyelid.

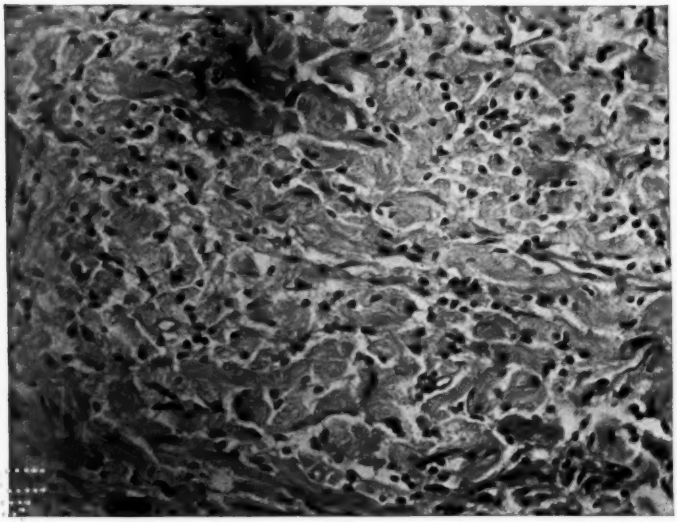

FIG. 2.—The Tumor (*Neurofibroma*) at the Apex of the Orbit.

REPORT OF THE REMOVAL OF A TUMOR AT THE APEX OF THE ORBIT, WITH PRESERVATION OF THE EYEBALL, IN A CASE OF PLEXIFORM NEUROMA OF THE EYELID. MICROSCOPICAL EXAMINATION.[1]

By Dr. ARNOLD KNAPP, New York.

(With two illustrations on Text-Plate XXV.)

The patient's previous history is that at the age of 9 months, presumably after a fall, a swelling appeared in the right upper lid; she was operated upon for this swelling by different ophthalmic surgeons at the age of 2½, 5, 12, and 15 years respectively. At the beginning of 1912, when the patient first came under my observation, she was a normally developed though mentally backward girl of 17 years with a number of round, pigmented spots on the back and legs.

The upper right lid was thickened, hung down, and could not be raised; there were irregular scars in the skin of the upper lid which extended to the temple. The eye was normal, proptosed, and there was a corneal scar. A tumor of the orbit was presupposed and an exploratory operation recommended.

Operation: January 5, 1912. After throwing the outer bony wall of the orbit backward as in the Krönlein operation, a horizontal incision was made in the periosteum above the level of the external rectus muscle. The index finger was carefully inserted and the contents and walls of the orbit explored; a round, elongated tumor was felt at the apex of the orbit just underneath the optic nerve. A path was carefully dissected in this direction and with the aid of blunt retractors the tumor was exposed and found adherent to the sheath of the optic nerve. This adhesion was separated,

[1] Presented at the meeting of the American Ophthalmological Society, Washington, May, 1916.

a capsule was isolated and opened and the tumor removed. The wound was closed with sutures after replacing the bone flap. The eyelids were sutured together to prevent the chemosis and lagophthalmos which are apt to follow orbital exploratory operations. Recovery uneventful.

The tumor measured *22 x 12mm.* It was soft; the cut section looked like a myxomatous gland. Microscopically it showed the structure of a neurofibroma (Figure 2); at the periphery normal and degenerated medullated nerve fibers were present, beautifully shown in sections which Dr. F. H. Verhoeff was kind enough to stain with phosphotungstic acid hæmatoxylin.[1] As one of the previous operations had been performed at the old hospital, some of the tumor which was removed from the eyelid was available and new sections were stained by Dr. Verhoeff; these showed the centrally placed medullated nerve-fibers and the other characteristics of the main growth as it occurs in the eyelid (Figure 1).

Plexiform neuroma is a well-defined though unusual tumor. Its frequent presence in the eyelid and adjoining temporal and frontal regions has brought it particularly to the attention of the oculist, and one of the earliest reports is by de Schweinitz (*T. A. O. S.*, vol. xxvii., 1891) before this Society twenty-five years ago. Alexis Thomson (*Neuroma and Neurofibromatosis*, Edinburgh, 1900) believes that plexiform neuroma must be included in the group of diffuse or generalized neurofibromatosis for it is the result of a localized fibromatosis following the distribution of a particular nerve or a nerve plexus. The two conditions may be met with in the same individual. He has collected sixty-two cases. Of fifty-eight cases, eighteen occurred in the temple, forehead, and upper eyelid, fourteen in the posterior part of the neck and behind the auricle.

Michel (*Graefe-Saemisch*, II. ed.) states that the distribution of this tumor corresponds to the distribution of the first branch of the V. nerve, though there is no pain as a rule. Aubineau and Civel (*Arch. d'opht.*, xxxi., p. 808, 1911) describe plexiform neuroma as having a triad of symptoms, consisting of cutaneous tumors, tumors of nerves, and pigmented spots, with

[1] Verhoeff's improved elastic tissue:
Stain with double quantity of ferric chlorid, 24 hours.
Rinse in water.
Mallory's phosphotungstic acid hæmatoxylin, 24 hours.
Rinse in water.
Alcohol, oil (if celloidin section), balsam.

general functional disturbances and arrest of development. These three principal symptoms were all present in our patient. The main or cutaneous tumor was situated, as is usual, in the upper lid and in the adjoining temporal region. The pigment spots on the skin of the back and legs and the retarded mental development are not unusual symptoms.

In addition to thickening of the skin of the half of the face (elephantiasis) and involvement of the bones of the face, the orbital complications are of particular interest. The tumor sometimes extends into the orbit. Sachsalber (Deutschmann's *Beiträge*, xxvii., 1898) described a plexiform neuroma of the orbit with buphthalmos. The growth was made up of coils of enlarged and varicose nerves lying in a mass of fibrous tissue. The association with buphthalmos has been noted several times. In a case of Snell and Treacher Collins (*T. O. S.*, 1903) the ciliary nerves were affected in their passage through the sclera. Rockliffe and Parsons (*Trans. Path. Soc.*, 1904) reported on a case where the entire orbit was filled with a mass consisting of nerves thickened and tortuous from fibromatosis. Beard and Brown (ARCH. OF OPHTH., vol. xxxv., 1906) described an orbital neuroma involving the supraorbital nerve; and Tertsch (*Graefe's Archiv*, 1903) observed a case of orbital tumor in which the lacrimal nerve was probably affected.

A complication of great interest are the tumors of the nerves. Thomson found in forty-five cases of generalized neurofibromatosis the cranial nerves affected in about two-thirds, the vagus in twenty-nine, and the trigeminal in twelve. According to Marx (*Z. f. A.*, p. 528, 1908), tumors have been described in the III., IV., and supraorbital branch of the V. nerves. Fehr (*Centralblatt f. prakt. Augenhlk.*, p. 233, 1913) reports on the case of a man with multiple skin tumors who showed a hemorrhagic neuroretinitis and a central scotoma in one eye. This developed into thrombosis of the central retinal vein and a diagnosis of neurofibroma of the optic nerve was made principally by exclusion, though an exploratory orbital operation revealed nothing.

In Aubineau and Civel's case there was, in addition to the tumor of the upper eyelid, a paralysis of the external rectus of that side. These authors collected six cases of intracranial localization from the French literature alone.

The case which I had the opportunity of observing was remarkable for the presence of a neurofibroma at the apex of the orbit, in addition to a plexiform neuroma in the lid. It cannot be stated with certainty to what particular nerve this tumor at the apex of the orbit was related; it probably was the nasal branch of the ophthalmic nerve, as this is the only sensory nerve just below the optic nerve in this position. At operation it was found closely adherent to the optic nerve sheath.

IMPROVED CAPSULE FORCEPS FOR INTRACAPSULAR CATARACT EXTRACTIONS.

By Dr. F. H. VERHOEFF, Boston.

(With one figure in the text.)

TWO years ago Arnold Knapp described before this Society a new method of intracapsular cataract extraction which he and Stanculeanu had independently devised. In this method the lens capsule was grasped with Kalt forceps and the lens dislocated by moving it from side to side, after which the forceps were removed and the lens expressed as in the Smith method. About a year ago I was informed that Török had modified Knapp's method in the following way: Instead of dislocating the lens he retained hold of the latter with the forceps and gently pulled it out while at the same time he made pressure on the cornea in the usual manner. Török's modification impressed me so favorably that I immediately tried it and have since continued to use it in almost all of my extractions of senile cataract.[1]

I have found that the method is most successful in cases of immature and hypermature cataracts; in the former evidently because the capsule is not degenerated, and in the latter because the capsule has become thickened and the zonule weakened. Lenses showing diffuse central haziness without marked peripheral striæ are especially suitable for the operation. Congenital cataracts in adults over thirty years of age I have also found may be safely removed by this method, for although

[1] Since this was written I find that Török dislocates the lens by Knapp's method before withdrawing it. Török's method is therefore quite different from mine. I am surprised to learn also that Török does not regard his method suitable for immature cataracts.

the zonule is apt to be resistant the capsule is still more so. According to Smith, congenital cataracts are not suitable for his method of intracapsular extraction.

In regard to the question as to the advisability of adopting the method as a routine procedure in all cases of mature senile cataract I have not yet come to a definite opinion. In some cases of mature cataract the lens comes away readily with the capsule intact, but in many the capsule ruptures, and in these cases I think the danger of loss of vitreous is somewhat increased. In the only case of intumescent cataract in which I tried the method, the lens came out intact readily enough but was followed by a slight amount of vitreous. In cases of immature cataract the danger of loss of vitreous is negligible, especially when compared with the advantages to be gained by removing the lens in capsule.

Whether or not I have carried out the operation in all its details in the same way as Török I do not know, since previous to this meeting he has not published a description of his method. I have found it necessary to make the incision only slightly larger than that for the ordinary method, since the lens tends to mold itself to the opening. It is important, I think, to grasp the capsule as low down as possible, so that when the lens is withdrawn traction will be made upon only a few fibers of the zonule at a time and less resistance thus encountered. For this reason as well as others, I dilate the pupil with atropine previous to the operation. After the capsule is grasped, the first movement should be to bring the lens directly forward toward the cornea, then it should be gently pulled out while deep pressure is being made along its lower edge through the cornea by means of the point of a large strabismus hook.

In most of my cases the operation was performed with iridectomy and without a conjunctival flap. During the past six months, however, I have employed a new method of wound closure which has enabled me safely to leave a round pupil. This method, which will be more fully described later, is briefly as follows: Before the cataract incision is made, a small quadrilateral conjunctival flap is dissected up above the cornea. A double armed suture is then passed superficially through the sclera for about 1*mm* parallel to and close to the

limbus beneath the flap. Each needle is then passed through the flap, leaving two loops of thread to allow room for passage of the lens. The two loops are then drawn to the outer side so that the cataract knife cannot cut them. An ordinary cataract incision is now made through the limbus, care being taken to bring the knife out beneath the conjunctival flap and in front of the scleral suture. If for any reason the anterior chamber is lost while the incision is being made, and the iris prolapses in front of the knife, the latter should be allowed to remain in position and the chamber refilled by inserting the tip of the irrigator into the incision along the blade. The lens is then removed as described, the iris partly replaced, and the suture firmly tied with a surgeon's knot. The iris is now completely replaced and then two very small peripheral button holes are made in it, one on each side of the conjunctival flap. When the capsule ruptures, or in case of an ordinary extraction, cortical matter is removed by irrigation after the suture is tied. The closure of the wound is so perfect that on one occasion I permanently restored the anterior chamber by means of the irrigator immediately after the operation. It probably would not be wise to do this in all cases, however, because should the patient break open the wound the sudden gush of aqueous might cause prolapse of the iris. This method of wound closure I believe to be an almost certain safeguard against infection subsequent to the operation.

After having had considerable experience with the Kalt forceps in intracapsular extractions it occurred to me that they could be improved in several important respects. In the first place, I found that it was not an easy matter to open the blades just to the proper extent and at the same time make the correct amount of backward pressure in the act of grasping the capsule. This difficulty I have overcome by means of a stop which allows the forceps to open only to the proper distance, about 2.5*mm*. This stop also acts in such a way when the forceps are closed as to limit the amount of pinching pressure to which the

capsule can be subjected. In other words, with this modification the only delicacy of manipulation required in grasping the capsule is in making the backward pressure upon the lens. Another serious defect in the Kalt forceps is that they are especially narrow just at the place where they must be held with the fingers. This, together with the facts that they are unduly top-heavy and not corrugated, makes them difficult to manipulate. I found also that the jaws of the Kalt forceps were bent at far too great an angle. These defects I have attempted to overcome in the forceps here illustrated, and in addition I have modified the biting ends of the instrument. Another improvement consists in the fact that the blades of the new forceps can come in contact only at the biting ends and at the heels, a space being left between these points which, in case of a simple extraction, renders it impossible for the iris to be caught. The instrument was made for me by Codman & Shurtleff of Boston.

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By LEWIS W. CRIGLER, M.D., Secretary.

MONDAY EVENING, APRIL 17, 1916. GEORGE H. BELL, M.D., CHAIRMAN.

Dr. W. H. Haskins read a paper on **Eye diseases resulting from dental lesions.** He said that it was very difficult to trace the actual relationship of dental lesions to many systemic and local conditions, but that the rapid improvement following removal of dental foci of infection was very strong presumptive evidence that the diseased teeth were at the bottom of the trouble. He spoke of the two ways in which the body suffers from the presence of septic foci; first by the absorption of toxines, and second by the invasion of bacteria. Investigation has shown that the streptococcus viridans is present in over 90% of mouths affected with diseased teeth or tonsils. This organism, when cultured and injected intravenously into rabbits, has been known to produce endocarditis, nephritis, iritis, articular affections, and many other systemic conditions. He said that positive bacterial cultures have seldom been obtained from the blood or fluids in these cases; as long as the patient's resistance is strong enough, only toxæmia may result. He thinks they (positive blood cultures) would be more often found if daily cultures were made over a period of time. He referred to Dr. Libman's investigations at Mt. Sinai Hospital, who has determined that many tissues of the body may harbor bacteria although they can not be demonstrated in the blood. He thinks the profession in this country backward in recognizing the above conditions, in view of the fact that it has occupied a prominent place in European literature for several

years. He quoted Kenneth Campbell as denying the existence of true rheumatic iritis. Worth states that more than 50% of cases of iritis, are due to oral sepsis and kindred conditions. B. T. Lang states that out of 176 cases of iritis seen by him, seventy-one were caused by mouth infections. Wm. Lang reports that out of 215 cases attributed to sepsis, 139 were traced to pyorrhea or other dental lesions, where only three were traced to the tonsils and two to the nose. De Schweinitz thinks that every case of uveitis is of septic origin, and hence he no longer uses the term rheumatic iritis.

Dr. Haskins enumerated the results in seven cases that had been referred to him for treatment. In six cases the eye conditions (iritis, two cases; iridocyclitis, three cases; keratitis, one) rapidly recovered, following the removal of the pus foci at the roots of the teeth. In one case the tonsils were very badly diseased; the patient had lost all of her teeth several years previously from pyorrhea. Her iritis promptly subsided, following the removal of her diseased tonsils.

Dr. Haskins then illustrated his paper with X-ray plates thrown on the screen, showing the condition of the teeth in several of the cases reported. By means of these pictures he showed how utterly impossible it is for surgeon or dentist to determine the condition at the roots of the teeth except by means of the X-ray. He condemns the gold crown and fixed bridge as harbingers of germs. Improper and imperfect filling of root canals is shown to be the rule and often the cause of abscess formation.

Several pathological specimens were shown illustrating the points of the paper.

Dr. J. G. DWYER discussed the **bacteriological relationship between diseases of the mouth and eye infections.** He said that this was a subject of particular interest to eye men, and that there were two means of determining this relationship. First, by means of the complement fixation test, and second, by means of blood cultures. He said that blood cultures after trauma were, as a rule, positive, but that the bacteria soon become destroyed if resistance is normal, and the infection continues to be a local one. He said that alveolar abscesses were typical examples of this type of infection. He differentiated between bacteræmia and septicæmia, the former being a

trivial affair and the latter very serious. Septicæmia being characterized by actual growth and multiplication of bacteria in the blood stream, whereas in bacteræmia the germs may gain entrance to the blood stream but their growth and multiplication are inhibited, and finally they become destroyed. Therefore unless the blood stream is constantly fed with bacteria from some focus of infection, bacteræmia does not mean very much. Dr. Dwyer thinks that if blood cultures were taken more often this condition would be better understood and lose many of its terrors. Dr. Dwyer said that he did not regard the complement fixation test as reliable in comparison with blood cultures.

Dr. Dwyer thinks that bacteria may invade the eye from abscesses around the teeth, just as mastoiditis may be followed by pyæmia with abscesses in the various joints, and compares the capillary plexuses about the joints with those in the interior of the eye as being quite similar in their anatomical arrangement; thus being predisposed to the same source of infection.

Dr. Dwyer laid stress on the fact that vaccines are of little value unless the focus of infection be removed.

Dr. JOSEPH M. LEVY, D.D.S., who was invited to discuss the subject from the dentist's standpoint said that he had little to add to what the previous speakers had said. He said that few dentists realized the importance of X-ray work in the examination and treatment of dental affections and for that reason not two per cent. of root canals were properly filled. He also showed X-ray pictures illustrating how utterly impossible it is for the dentist to do his work properly without the aid of the radiograph, and thus, work improperly done, becomes the starting point of abscesses such as here described.

The discussion was entered into by Drs. MARPLE, CLAIBORNE, TYSON, WOOTTON, DAVIS, and SCHIRMER; all agreeing that the old term "rheumatic iritis" must be relegated to the past as soon as our knowledge can be made a little more definite as to the ætiology of this affection; that oral sepsis (and especially alveolar abscesses) either by their toxines or bacteria which enter the blood stream are responsible for a large percentage of the cases of iritis and iridocyclitis, heretofore classified as rheuma ic.

Dr. SCHIRMER thinks that bacterial invasion of the eye in

these cases is to be excluded until proven, since it is not likely that, once they gain entrance to the eye, removing the primary source of infection will do any good. He thinks that the condition is one of toxæmia.

Dr. Dwyer said that he thought that clinical evidence should count for something and that it was not impossible for germs to enter the eye and be destroyed, just as they are in other parts of the body, provided that they are not too virulent and not in overwhelming numbers. He does not doubt but that toxæmia plays a large part in these conditions.

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

Annual Congress.

By C. DEVEREUX MARSHALL, F.R.C.S., London.

The Annual Congress of the Society was held on the 4th, 5th, and 6th of May, under the presidency of Mr. Walter H. Jessop, F. R. C. S. The discussions took place at the Home of the Royal Society of Medicine, and the clinical meeting at the Central London Ophthalmic Hospital, Judd Street.

Presidential Address.

The President, in the course of his opening address, referred with pride to the fact that the Society was now presenting its thirty-sixth consecutive annual volume of Transactions. The Bowman Library was the finest and most complete Ophthalmological library in the world, and he asked members to co-operate in keeping it supplied with literature of special interest to their department. This year the Society was made poorer by the death of two brilliant workers: Mr. George Coats, of London, and Professor Straub, of Amsterdam. A special tribute was due to the first named for the great help he had rendered to the Society, especially in its troublous times three years ago. Mr. Jessop devoted the chief part of his address to the subject of some ophthalmic lessons of the war. Thanks to the efforts of Colonel Lister and others, the treatment of cases of eye injury had been good. He, the speaker, had, as yet, found no cases of sympathetic ophthalmitis, which happy fact was due to the precaution of removing every por-

tion of a smashed and broken eye. In the war of 1870, sympathetic eye trouble was reported in 55.6% of cases of severe eye injury. This war had taught ophthalmic surgeons much on the subject of papillœdema, a question which had been patiently worked at by Mr. Leslie Paton and Dr. Gordon Holmes. He proceeded to refer to trench nephritis and the eye changes found in association with it. He regarded it as a toxic condition, and referred to the occurrence of œdema of the retina, ocular hemorrhages, and the formation of white plaques. In the worst instances, the blood pressure was high, but it varied much in different cases. In encouraging younger ophthalmologists to accurate observation, he laid stress on the importance of daylight examination.

On the motion of Sir Anderson Critchett, the President was cordially thanked for his address.

Mr. J. HERBERT FISHER read a paper on **Leber's disease (hereditary optic atrophy): a suggestion as to its cause.** He suggested that the causal agent might be a disturbance of the pituitary body, of temporary duration, and moderate in degree, implicating the visual pathway. If his view were correct, organo-therapy ought to be beneficial. During pregnancy the pituitary gland might enlarge to two or three times the normal, and there was a close relationship between the pituitary and sexual glands. Moreover, the pituitary and optic chiasma were in such close contiguity that any small swelling of the former would extend to and make itself felt in the latter. He exhibited a skiagram showing a honeycomb-like shadow in the situation of the pituitary.

The paper was discussed by Sir GEORGE BERRY, Dr. G. MACKAY, Dr. S. A. K. WILSON, Mr. STACK, Mr. STEPHEN MAYOU, and Dr. C. O. HAWTHORNE.

Mr. FOSTER MOORE contributed a paper on **The retinal circulation in arteriosclerosis.** He agreed with Priestley Smith that a high pressure in accessible arteries in elderly men did not necessarily mean high pressure in the ciliary capillaries. A sudden obstruction of circulation due to thrombosis in an artery might lead to relative starvation of tissues, and the patient might awake to find himself blind. Sometimes there was an equally rapid recovery of vision. It was not necessary to evoke spasm of the artery as a cause.

Dr. S. A. K. WILSON read a paper on **Dysmetropsia,** meaning thereby a defect in the capacity to appreciate the size and distance of objects. He divided the cases into (*a*) those in which the lesion was presumably peripheral; (*b*) those in which it was probably central; (*c*) psychical cases. In an instance of the latter, the young woman, who was the subject of hysteria, always saw the right half of objects much larger than the left: and if two shillings were placed side by side, she considered that the right hand one was half-a-crown. In a case of occipito-parietal tumor, the objects looked at seemed so near that they appeared to be practically touching the observer's face. Dr. Wilson considered that in judging of distances and sizes of objects we learn more from the retina than from so-called kinæsthetic impressions.

Mr. M. HINE recorded some observations with the Schiötz tonometer, as a result of which he was of opinion that eserine produced no injury, although many of the cases observed were in children; and that it definitely reduced intraocular tension, a view shared by a number of workers who had recorded their observations.

Mr. E. TREACHER COLLINS read a paper on **Contusion hypotony.** He said that diminution of tension after contusion of the eye, apart from perforation of the globe, was not infrequent. In most cases, normal tension was restored in a few days: in others not for several weeks, and in some it remained so long as to be likely to be permanent. The causes he discussed under the following heads:

(1) Diminished secretion, cases being divisible into (*a*) from nerve inhibition; (*b*) from vascular disturbance; (*c*) from epithelial damage.

(2) Increased excretion, namely (*a*) through normal channels; (*b*) through newly-formed channels. He submitted the following conclusions:

1. Hypotony following contusion of the eyeball may be due to different causes, more than one of which may be present at the same time.

2. When of short duration it is probably due to an increased rate of excretion of the intraocular fluid through the expanded normal channels or exit, or possibly to some arrest of secretion, from paresis of the vaso-constrictor nerves.

3. When of long duration, it may be due to (*a*) the formation of new channels of exit for the intraocular fluid from the anterior chamber, from either an internal scleral rupture, or rupture of the pectinate ligament; (*b*) the cutting off of blood supply to the ciliary body from rupture of the anterior ciliary arteries, or (*c*) possibly the detachment of the pars ciliaris retinæ.

4. If accompanied by extensive hemorrhage into the anterior chamber, either the canal of Schlemm has been opened up by an internal scleral puncture, or the anterior ciliary arteries have been torn across from cyclodialysis.

5. If, when the blood has been cleared away, a portion of the iris has disappeared from view, as though an iridectomy had been done, then there has been cyclodialysis with rupture of the anterior ciliary arteries.

6. If, in the course of time, a translucent area appears just outside the sclero-corneal margin, like that seen in cystoid cicatrix, then there has been an incomplete internal rupture.

7. If the anterior chamber, without extensive hemorrhage into it, becomes markedly deepened in the whole or part of its circumference, there has probably been a rupture of the ligamentum pectinatum, limited to the pillars of the iris, and of the ciliary muscle, prolonging the angle of the anterior chamber outwards.

8. If the lens is dislocated laterally and the retina detached, the vitreous tumor has probably come forwards into the circumlental space, and may have dragged the pars ciliaris retinæ away from the pigment epithelium.

Mr. A. L. WHITEHEAD (Leeds) and Mr. J. HERBERT PARSONS (London) introduced a discussion on **foreign bodies in the eye and orbit.**

Mr. WHITEHEAD referred to the frequency with which the eyeball was penetrated by foreign substances in industrial centers. Sometimes the particle passed completely through the globe, and the war had demonstrated the extraordinary penetrating power of metals when propelled by high explosives. He enumerated the various forms of eye injury, and said that the prognosis and treatment of an eye containing a foreign body must depend on a variety of factors, such as the introduction of septic organisms with the foreign body, the size

of the substance, the site and depth of penetration, the nature of the substance. Copper, unless embedded in the lens, always set up localized suppuration, with necrosis of tissue, and, if in the vitreous, phthisis bulbi. He discussed X-ray localization, and the extraction of steel and iron by the electro-magnet. If steel were in the vitreous, he made a conjunctival flap, punctured the sclerotic behind the ciliary region, and inserted the point of the giant magnet, keeping the current on during withdrawal. In his hospital, the onset of sympathetic iridocyclitis secondary to retention of a foreign body inside the exciting eye was not common. An injured eye without useful vision should be enucleated if there was even a suspicion of the presence in it of a foreign body. If a foreign substance were not causing pain or discomfort, it should be left alone.

Mr. PARSONS discussed the anatomical relations of the eyeball to the orbit, and urged a more accurate study of the relations of the living eye to the bones, which could be done by the insertion of slips of lead foil in the cocainized conjunctival sac. The eye was least protected against missiles coming from a direction downwards and outwards. Despite the heat generated in a high-velocity missile, the particles entering the eye were not always sterile. The diagnosis, pathological condition, treatment, and prognosis all depended profoundly on the nature of the foreign body. When the substance could not be seen in the eye, skiagraphy must be employed, but this had its severe limitations. He regarded accurate localization as most important. If this were not done within 1*mm* of the truth it might mean the needless sacrifice of the eye. Only rarely did a copper particle become encapsulated, with restoration of useful vision. He discussed at some length the production of siderosis bulbi. Glass and porcelain might cause remarkably little reaction in the eye, but eventually the eye became disorganized following iridocyclitis. The lens substance and the vitreous formed excellent culture media. The ultimate results of extraction of intraocular foreign bodies by the electromagnet were of profound importance, and had not yet received their due attention. In conclusion, he alluded to the different operative measures, according to the class of case, and the prognosis in them.

There was a very instructive discussion, and the authors replied.

A debate on **the treatment of the syphilitic eye affections by the newer methods,** was introduced by Mr. J. B. LAWFORD and Captain H. S. BROWNING.

Mr. LAWFORD opened with a historical review of the subject. When salvarsan was introduced, sleeping sickness had already been treated by atoxyl, and, in spite of the cases of blindness which followed its use, it was extensively prescribed for patients suffering from ocular syphilis. The frequency of occurrence of nerve atrophy afterwards, however, soon led to the abandonment of those drugs. Salvarsan was a much less dangerous arsenic compound. He suggested that as there was now a very general agreement as to the value and efficiency of salvarsan therapy in ocular lesions in the early periods of syphilis—primary, secondary, and early tertiary stages (*i.e.*, chancre of eyelids, iritis, cyclitis, etc.), a discussion on this aspect from the clinical side would not be very helpful. Much difference of opinion, however, existed as to the efficacy of salvarsan in some of the later manifestations, and on this side discussion would be valuable. He spoke of the use of salvarsan in oculomotor paralysis, ophthalmoplegia interna, and optic nerve atrophy of late syphilis, and in congenital keratitis, and quoted cases, both from his own experience and the published records. He concluded with a brief reference to the reputed noxious effects of salvarsan and neo-salvarsan upon the optic and other ocular nerves, and said most observers were agreed that the complications met with after the use of these drugs were really fresh manifestations of the syphilitic virus, and its continued employment resulted in their amelioration, differing therein from the nerve lesions following the use of atoxyl. He believed there was no evidence that syphilis could be eradicated by salvarsan treatment alone; prolonged employment of the classical remedies was essential for a cure, even when the results from the arsenic compounds seemed to be brilliant.

Captain BROWNING dealt first with the diagnosis of the disease, which was too frequently based on clinical evidence alone. All aids to diagnosis should, however, be employed. It was not true that all cases of choroiditis were syphilitic in

origin. In every suspected case of ocular syphilis, a careful examination of the whole body should be made, and the lesions examined with the dark ground condenser. He preferred the original Wassermann test for ordinary purposes, and he considered it one of the most reliable serological tests. A negative Wassermann, however, did not mean that the patient had not got syphilis. Repeated doses of arsenical preparations, combined with mercurial treatment, were needed to bring about a cure. He spoke of the value of the various drugs for the disease, and their dosage and methods of administration. Kharsivan and neokharsivan were more toxic than salvarsan, and there were more unpleasant symptoms after them. He was not encouraged by his trials of intramine and ferrivine to continue their use.

The debate, which became general, revealed a strong agreement with Captain Browning's contentions.

On Friday afternoon the Society examined and discussed a large series of cases of special interest at the Central London Ophthalmic Hospital, Judd Street.

On Saturday, Major CUNNINGHAM, Professor DE LAPERSONNE, and Colonel LISTER discussed war injuries to the eyes. The last-named said that in the case of foreign bodies in the eye, so long as there was no prolapse of uvea or lens capsule, there seemed to be no danger in keeping that eye. The lens had been frequently injured in the war. The absence of cases of sympathetic ophthalmia from the war was extraordinarily satisfactory. For cases of prolapse of the uvea, he submitted a new operation, and emphasized the importance of avoiding tension, if necessary, by making a counter-puncture. If an eye containing a foreign body did not quiet down, their practice in France was to remove the eye. But if it did become quiet, the eye was left in for colleagues in England to treat and watch it He invited criticism from ophthalmologists in this country of the work in France in eye cases, so that workers in the war area might have the benefit of such suggestions.

The following papers were read:

Dr. C. O. HAWTHORNE, **Retinal changes in glycosuria.**

Captain A. W. ORMOND, **Treatment of large traumatic hemorrhages in the vitreous.**

Mr. J. GRAY CLEGG, **Spontaneous intraocular hemorrhages.**

Captain BEATSON HIRD, **Notes on a case of tumor of the retina.** The case was regarded as unique, and was referred, with specimens, to a pathological committee to report upon it.

QUARTERLY REVIEW OF THE PROGRESS OF OPHTHALMOLOGY.

By H. KOELLNER, Berlin; W. KRAUSS, Marburg; R. KÜMMELL, Erlangen; W. LOEHLEIN, Greifswald; H. MEYER, Brandenburg; W. NICOLAI, Berlin; H. PAGENSTECHER, Strassburg; K. WESSELY, Würzburg; and M. WOLFRUM, Leipsic, with the Assistance of Drs. ALLING, New Haven; CALDERARO, Rome; CAUSÉ, Mayence; CURRAN, Kansas City; DANIS, Brussels; GILBERT, Munich; GROENHOLM, Helsingfors; v. POPPEN Petrograd; TREUTLER, Dresden; and VISSER, Amsterdam.

Edited by MATTHIAS LANCKTON FOSTER, M.D., F.A.C.S., New Rochelle, N. Y.

FOURTH QUARTER, 1915.

I.—GENERAL OPHTHALMIC LITERATURE.

1. AYER, J. B. **Lumbar puncture and examination of the spinal fluid in affections of the eye.** *Transactions American Ophthalmological Society*, 1915.

2. COHN. **The blind man as a counselor to the blind and with regard to their care.** *Vortrag i. d. Schlesischen Gesellsch. f. vaterl. Kultur*, Dec. 3, 1915.

3. FRIEDENWALD, H. **The ophthalmias of Palestine.** *Transactions of the American Ophthalmological Society*, 1915.

4. GAMBLE, W. E. **Albinism in man with special reference to the eye.** *Annals of Ophthalmology*, October, 1915.

COHN (2, **The blind as a counselor to the blind**), who became blind himself at 6 years of age, urges that the blind victims of the war be brought together as soon as possible after the end of their medical treatment into institutions for the blind, where they may be taught some trade in accordance with their ability. In addition to the trades customarily taught the blind he suggests a number of others, including cigar maker, packer, and polisher.

AYER'S (1, **Lumbar puncture and examination of the spinal fluid in affections of the eye**) paper is based upon the spinal fluid examinations in 160 cases of interest to the ophthalmologist. There were forty cases of external ocular palsy and in all but five there was evidence of meningeal irritation as demonstrated by increase in the number of cells, increased proteid, and positive Wassermann. When the palsy is due to active syphilis, both blood and spinal-fluid tests are usually positive, but the latter is the more reliable. In forty-four cases of primary optic atrophy only twelve had normal spinal fluid, but in four of these the test was made after antisyphilitic treatment. If the case is well advanced he thinks the outlook absolutely bad, but when taken early and if slowly progressive salvarsan offers some chance. Regarding optic neuritis he cautions against spinal puncture when choked disk is present as many cases of sudden death are recorded. He thinks that the Wassermann is fortunately usually to be relied upon in optic neuritis. Concerning the pupil, it is the exception to find an advanced case of nerve syphilis with normal pupils. If Argyll-Robertson pupils are found without other symptoms the case is usually very puzzling. He has seen thirteen such cases. The spinal test is important as indicating when treatment is to be pushed. Pupils irregular in the two eyes are very often the result of syphilis, but whether or not should be left to the test.

ALLING.

FRIEDENWALD (3, **The ophthalmias of Palestine**) has made two visits to Egypt and Palestine and given a description of his studies of affections of the conjunctiva in the latter country. Simple conjunctivitis is very common and due to the bright light and dust. Phlyctenular ophthalmia is comparatively rare. Morax-Axenfeld conjunctivitis is of frequent occurrence and generally associated with trachoma. The Koch-Weeks bacillus is the common cause of acute conjunctivitis. Gonococcic ophthalmia neonatorum was not observed. Trachoma is not found in Palestine in such large percentage as in Egypt, where from 90% to 95% of school children are said to be infected, although in some localities it may approach these figures. In children there is a relative absence of corneal lid and lacrimal complications and it responds readily to treatment.

ALLING.

GAMBLE (4, **Albinism in man with special reference to the eye**) states that in the albinotic eye the lack of pigment is most marked and characteristic in the retinal pigment, causing reduction of vision and nystagmus. Microscopic examination of these eyes has been made only six times. Four cases of albinism in the eyes without involvement of the hair or skin have been reported. The condition is undoubtedly of hereditary origin.

ALLING.

II.—RELATIONS OF OPHTHALMIC TO GENERAL DISEASES, INCLUDING POISONS.

5. BLACK, A. I. **Ocular diseases resulting from dental lesions.** *Ophthalmic Record*, December, 1915.

6. GREEN, JOHN, Jr. **Ocular study of a case of leontiasis ossea, with a synopsis of reported cases showing involvement of the eyes and adnexa.** *Amer. Jour. of Ophthalm.*, October, 1915.

7. HESS, OTTO. **Specific treatment of pneumonia with optochin (ethylhydrocuprein).** *Münchener med. Wochenschr.*, 1915, p. 1528.

8. HIPPEL, E. V. **Studies of the pathology of the thymus gland with the aid of Abderhalden's method.** *Fermentforschung*, vol. i., p. 233.

9. JOSEFSON, A. **Disturbances in the visual fields in cases of tumor of the hypophysis, with especial reference to bitemporal hemianopsia.** *Klinische Monatsblaetter f. Augenheilk.*, Nov.-Dec., 1915, p. 636.

10. MCMURRY, S. **Some ocular effects of plumbism.** *Ophthalmoscope*, Nov. 1, 1915.

11. WOODS, HIRAM. **Ocular phenomena accompanying three cases of gastrointestinal disorder.** *Trans. American Ophthalmological Society*, 1915.

BLACK (5, **Ocular diseases resulting from dental lesions**) divides the ocular disturbances which result from dental lesions into two groups: reflex neuroses which may be sensory, vasomotor, or muscular affections, and inflammations of various tissues of the eye due to infection. The former are always caused by irritation of the vital pulp of the tooth by thermal changes or contact of a foreign body with the exposed pulp. The latter are practically always due to an infection involving the peridental membrane either in the form of an alveolar abscess at the root apex or a pus pocket alongside the root.

ALLING.

The questions to which answers were sought by VON HIPPEL (8, **Studies of the pathology of the thymus gland with the aid of Abderhalden's method**) were: Has the markedly frequent

exhaustion of the thymus an essentially different signification from the exhaustion of other organs? Is it or is it not contradictory to the theory of the specificity of protective ferments? What is its clinical importance? He comes to the following conclusions as the results of his studies: A clinically pathological condition may be present without necessitating the presence of a positive reaction with the organ in question. Likewise a positive serum reaction may be observed when the clinical condition is negative. The main fact is that, through the use of this procedure in certain groups of eye diseases, like glaucoma, diseases of the optic nerve, and senile cataract, a hitherto wholly unknown morbid general condition may be first discovered and established with absolutely astounding frequency, and that, further, in this group of cases a surprising agreement between the urological and clinical conditions can be proved. The greatest care is necessary in drawing conclusions for the clinic, as the reaction is perfectly compatible with the ordinary conception of health. A loss of function of the gland does not necessarily induce any observable symptoms. On the other hand, it is certain that the positive thymus reaction coincides in a very considerable part of the cases with the so-called status thymicus, or thymico-lymphaticus. Hence it is not right to consider the finding of this reaction to be of no importance, even though by itself it does not settle the diagnosis. If one will use the reaction regularly in the clinic, and when it is positive search with all clinical aids for the status thymico-lymphaticus, he will find the latter very often where it is now overlooked. It would therefore be of great value if such studies were instituted in other places on patients suffering from glaucoma and the other diseases mentioned. Indirect evidence could thus be obtained from an agreement or disagreement of the results for or against all connection of the disease of the eye with the general condition. The nature of the loss of function of the thymus in the cases in which no status thymicus exists is entirely beyond our knowledge, but it is certain that the loss frequently happens with no clinical evidences. Finally von Hippel says that by the administration of thymin, in spite of hyperplasia of the thymus, the positive reaction may be changed gradually into a negative.

Out of eighty-one cases of pneumonia treated with various

doses of optochin HESS (7, **Specific treatment of pneumonia with optochin**) observed disturbances of the eyes in eight. These disturbances consisted of flickering before the eyes, amblyopia, amaurosis, contractions of the visual field, pallor of the optic nerves, dilatation and immobility to light of the pupils. All of these with the exception of two passed away promptly after discontinuance of the remedy without any impairment of the vision. The worst cases occurred in two patients who had been treated with optochin from the second day of sickness, which does away with the idea that such harm is more likely to be done when the treatment is instituted in the later stages of the disease. The first ocular symptoms of trouble appeared after the ingestion of from two to nine grams. The worst cases were those in which 0.2 of the remedy was given eight times a day; in only one out of fifty-eight cases in which the same dose was given six times a day did any disturbance appear, and this was an extremely slight flickering; six doses a day of 0.25 brought on symptoms in two patients, but they passed off when the dose was reduced to 0.2.

JOSEFSON (9, **Disturbances in the visual fields in tumors of the hypophysis**) claims that in 1903 he described these disturbances, and that he at that time called attention to a quadrant hemianopsia or achromatopsia upwards and outwards as an important early symptom of enlargement of the hypophysis. He also claims to have been the first to demonstrate roentgenologically the enlargement of the sella turcica in acromegaly.

This disease is rare and characterized by either general or localized hyperostosis of the bones of the face and cranium. It begins in early life and is progressive although there may be periods of remission. The eye, ear, and nose are frequently affected. Exophthalmos, separation of the orbits, muscle paralysis, optic atrophy, and optic neuritis are among the ocular complications. GREEN'S (6, **Ocular study of leontiasis ossea**) patient showed a disfiguring enlargement of the upper part of the face and skull. Both globes were prominent and widely separated with limited motility. The optic disks were pale but the vision was $\frac{6}{6}$. During the eight years the patient was under observation the exophthalmos had appreciably

diminished and there was a marked decrease in the bony hypertrophy.

ALLING.

McMURRY (10, **Some ocular effects of plumbism**) has been able to follow up sixteen cases of plumbism. All showed at one time or other contraction on the field of vision from five to twenty-five degrees. The contraction seemed to have an intimate connection with acuity of vision and degree of headache. Three showed a central scotoma for red and blue. Ten showed a contraction of the field for blue and, of these, three showed an actual interlacing of red and blue. There are no gross changes in the fundus excepting after severe headache or sickness. While at one time the choroidal vessels may be seen clearly, these may be masked the next day by slight œdema of the retina, and the field is then more contracted.

CURRAN.

WOODS'S (11, **Ocular phenomena accompanying three cases of gastrointestinal disorder**) three cases had one common feature—gastrointestinal stasis. In the first there was a chronic uveitis and it seemed possible that the primary focus of infection was in an unrecognized alveolar abscess, although the cause might also be attributed to the intestinal disorder. The second case, showed all the signs of toxic amblyopia. There was also intestinal stasis complicated by gall stones and occasional glycosuria. An attack of exudation in the retina was accompanied by all the signs of autointoxication. The third case, of constantly shifting paracentral scotomata, was very unusual. The association between the size and position of the scotomata with indiscretion in eating was suggestive.

ALLING.

III.—GENERAL AND EXPERIMENTAL PATHOLOGY AND TREATMENT.

12. BORDLEY, J. **Malignant uveitis treated with thyroid extract.** *Transactions of the American Ophthalmological Society*, 1915.

13. GOLDSCHMIDT. **Experimental contribution to the ætiology of keratomalacia.** *Archiv f. Ophthalmologie*, xc., p. 354.

14. GRADLE. **Some clinical experiences with optochin.** *Klinische Monatsblaetter f. Augenheilkunde*, September–October, 1915, p. 362.

15. KNAPP, A. **Salvarsan in interstitial keratitis and in optic-nerve lesions.** *Transactions of the American Ophthalmological Society*, 1915.

16. NIETSCH. **Treatment with lipoiodine in ophthalmic practice.** *Fortschr. d. Medizin*, 1., p. 1107.

17. OESTERREICHER. **Rhodalzid in ocular eczema.** *Zentralbl. f. prakt. Augenheilkunde*, November–December, 1915, p. 222.

18. VON SZILY. **Concerning the action upon the eye, and concerning the nature of the anaphylotoxin obtained from the bacillus prodigiosus, together with experiments concerning the inflammatory material circulating in rabbits' blood after incorporation of bacteria.** *Klinische Monatsblaetter f. Augenheilkunde*, September-October, 1915, p. 235

VON SZILY (18, **Anaphylotoxin from the bacillus prodigiosus**) has confirmed the experiments of Dodd and Rados and found that the bacillus prodigiosus is by no means an absolutely harmless saprophyte. There is an essential difference between the bacterial anaphylatoxine and the prodigiosus extract. The inflammation-exciting property of the latter depends on the effect of the extracting fluid on the cornea of the animal, on the circumstance that it varies in suitability for extraction, and on its content of extracted toxines, the last factor being the most important.

BORDLEY'S (12, **Malignant uveitis treated with thyroid extract**) cases were of the type showing photophobia and loss of vision, with exudates on the posterior surface of the cornea in the form of dots or plaques. There were also dilated pupils and discolored irides with vitreous opacities. In none of the eight cases was disease found in any of the vital organs, but they all gave a positive tuberculin reaction. He treated these cases with thyroid extract with marked success in four, doubtful results in two, and failure in two. In spite of these results he does not wish to leave the impression that malignant uveitis is necessarily a symptom of cretinism of myxœdema.

ALLING.

KNAPP'S (15, **Salvarsan in interstitial keratitis and in optic-nerve lesions**) method of treatment of interstitial keratitis is to give the patient Hg. by inunctions, small doses of KI, and neosalvarsan in five doses of one-half strength every three days, then after two weeks to give salvarsan in one-half doses every three days for five times. He thinks that the earlier the treatment is begun the better the results. In all cases inflammatory signs quickly subside. Salvarsan, however, does not prevent the other side from becoming affected. In syphilitic optic neuritis and atrophy due to tabes and paresis he

believes that salvarsan does not have any unfavorable action on the nerve. In atrophy, treatment must necessarily be preventive and limited to an attempt to keep the spinal fluid as normal as possible.

ALLING.

GOLDSCHMIDT (13, **Ætiology of keratomalacia**) succeeded in inducing the clinical and pathological picture of keratomalacia in white rats through partial underfeeding. The symptoms were then made to disappear by the addition to the food of *2ccm* of raw dry milk, the action of which does not seem to depend on the presence of albumin, fat, carbohydrates, or salts, but upon some as yet unknown substances that are necessary to life. It seems probable that this keratomalacia in the rats is to be ascribed to an experimental scurvy, or beriberi. Partial under-nutrition likewise plays an essential part in the production of human keratomalacia.

GRADLE (14, **Clinical experiences with optochin**) obtained good results from optochin, not only in the treatment of pneumococcus ulcer of the cornea, but also in twenty-six cases of pneumococcal conjunctivitis. In purulent dacryocystitis, the effect fluctuates a great deal, because the infection often is mixed. No benefit was obtained in trachoma, yet he got good results in two cases of xerosis.

NIETSCH (16, **Lipoiodine in ophthalmic practice**) considers that lipoiodine, a fatty acid derivate of iodine, possesses marked advantages over potassic iodide. While the latter remains in the body only a very short time, the lipoiodine is absorbed completely and excreted slowly. No iodism was observed, and no gastrointestinal disturbances, even after it had been used for months. He thinks it may be beneficial in many cases of interstitial keratitis, optic atrophy, and papillitis that are due to syphilis.

OESTERREICHER (17, **Rhodalzid in ocular eczema**) finds that almost all persons with eczematous keratoconjunctivitis suffer from dental caries. The following are his conclusions from a study of fifty cases: (1) Dental caries is a typical accompanying symptom of the exudative diathesis. (2) The saliva contains its normal content of rhodium in a third of the cases. When the rhodium is absent or in diminished quantity treatment with rhodalzid should be instituted, giving a tablet

twice a day, a half tablet to children. The medication should be continued for fourteen days, intermitted for eight days, then resumed for fourteen days. He thinks that through this means the course of the eye disease could be accelerated, and asks that the method be tested, as his own experiments are not yet concluded.

IV.—OPERATIVE TECHNIQUE.

19. HANSSEN. **The formation of a stump after enucleation or exenteration of the eye.** *Zeitschrift f. Augenheilkunde*, July-August, p. 89.

20. SALZMANN. **Ophthalmoscopy of the sinus of the anterior chamber.** *Ibid.*, p. 26.

21. SALZMANN. **Supplement to the above paper.** *Ibid.*, September, p. 160.

22. SCHLABS. **Measurements with Hertel's exophthalmometer.** *Klinische Monatsblaetter f. Augenheilkunde*, November-December, 1915, p. 611.

23. SCHOUTE. **Strengthening of the giant magnet.** *Zeitschrift f. Augenheilkunde*, July-August, 1915, p. 69.

24. SWEET, W. M. **Some experiences in enucleation under local anæsthesia.** *Transactions of the American Ophthalmological Society*, 1915.

25. TERRY, R. J., and WIENER, M. **The mydriatic action of dextrohyoscyamine.** *Annals of Ophthalmology*, October, 1915.

SALZMANN (20 and 21, **Ophthalmoscopy of the sinus of the anterior chamber**) describes the technic of this procedure, the normal sinus, malformations, and pathological changes to be found there. The conditions described include hemorrhage into the anterior chamber, siderosis, wounds in the region of the root of the iris, iritis and its sequelæ, glaucoma, hydrophthalmos, and keratoconus. In his supplement he admits that Trautas in Constantinople performed not only ophthalmoscopy of the ciliary body, by pressing his finger upon the globe so as to bring this body into view, but also of the sinus of the anterior chamber. Trautas published his observations in French in 1907. It may be mentioned also that Mihno got a view of the sinus of the chamber in 1913 by filling the conjunctival sac with water and having the patient look down.

SWEET (24, **Some experiences in enucleation under local anæsthesia**) has enucleated nine eyes by the method of Elschnig, which consists in injecting 1% cocaine with adrenalin, the needle entering below the external rectus and being pushed

into the orbit to about the situation of the ciliary ganglion. Four injections are then made at the insertions of the recti muscles. In none of the cases was there much pain, the only complaint being made when the muscles were drawn forward on the hook. He later enucleated in twelve cases by the method of Siegrist, using 1% novocaine with adrenalin at the insertion of the muscles and in the region of the optic nerve employing a curved needle. Nine of these operations were without pain.

ALLING.

TERRY and WIENER (25, **Mydriatic action of dextrohyoscyamine**) found that a one-half per cent. solution of this drug caused dilatation of the pupil with only a slight effect upon the accommodation. The mydriasis passed off in about twelve hours. Its advantage over euphthalmine is that it dilates the pupil more quickly. It is, however, unstable in solution and its instillation is accompanied by smarting.

ALLING.

SCHLABS (22, **Measurements with Hertel's exophthalmometer**) took measurements on fifty people standing, lying, and after stooping for five minutes. The resultant average fluctuated between 10.1 and 21*mm*. A conclusion from these measurements as to the physiological fluctuations in various positions of the head is not possible. He found that in the majority of cases the distance from the apex of the cornea to the lateral margin of the orbit was greater on the right side than on the left.

SCHOUTE (23, **Strengthening of the giant magnet**) finds that by using the alternating current a powerful electrolytic element is introduced.

HANSSEN (19, **Formation of a stump after enucleation**) says that neither the implantation of the bones of cattle, nor transplantation of fat or of cartilage gives satisfactory results, but that these may be obtained by means of transplantation of bone covered with periosteum. He uses for this purpose the head of a metatarsal or metacarpal bone.

V.—ANATOMY, EMBRYOLOGY, MALFORMATIONS.

26. BÖHM. Clinical and pathological studies of congenital partial aniridia. *Klin. Monatsbl. f. Augenheilk.*, lv., p. 544.

27. MÜLLER. **A second case of bilateral symmetrical lipodermoid at the inner canthus.** *Zeitschrift f. Augenheilkunde*, xxxiv., p. 157.

28. PETERS, R. **Congenital lagophthalmos in four generations.** *Klin. Monatsblätter f. Augenheilkunde*, lv., p. 308.

29. STEIGER. **Heredity in the human eye.** *Zeitschrift f. Augenheilkunde*, xxxiv., p. 1.

BÖHM (26, **Congenital partial aniridia**) studied anatomically an eye with congenital partial aniridia, better irideremia, that had suffered from secondary glaucoma. There was only a bridgelike piece of iris, but in one place in this a pupillary action could be obtained. The eyeball was enlarged as a whole. Where the iris was well developed the sinus of the anterior chamber was open and Schlemm's canal distinctly visible. In the iris a sphincter, and on depigmented sections a dilatator, was demonstrable. Where no iris could be perceived clinically a rudiment was present that simulated a ciliary process and contained no muscular tissue. In a series of sections where the stump of iris terminated, lamellæ of the cornea extended into the anterior chamber, as Descemet's membrane was lacking at this place. The cord was adherent to the anterior limiting layer of the vitreous, which here projected forward, so that one might speak of a hernia of the vitreous. This corneal cord is to be considered in Böhm's opinion as a primary malformation, a residuum of the mesoderm, and he ascribes to it a large part in the origin of the malformation of the iris, as with the disappearance of the cord from the sections the stump of iris became longer. Otherwise the conditions found agree in essentials with those found in other similar cases. At any rate in this case the ectoderm and mesoderm seem to have been involved in the embryological disturbance.

MÜLLER (27, **Bilateral symmetrical lipodermoid at the inner canthus**) reports a case of this nature which is claimed to be the second on record. The patient was a seventeen-year-old girl, in whom the caruncles were displaced equally, the upper punctum was absent on one side and led into a short cul-de-sac on the other. The tumors were removed and the diagnosis confirmed microscopically.

A congenital lagophthalmos present in great-grandmother, grandmother, mother, and child was seen by PETERS (28, **Congenital lagophthalmos in four generations**). The orbi-

cularis did not functionate properly, and in consequence of an absence of Horner's muscle, and a drooping of the lower lid, there was a considerable epiphora.

STEIGER (29, **Heredity in the human eye**) concludes from a large number of measurements of the refraction and of the cornea that heredity is responsible for a number of characteristics in the eye, all of which may be looked upon as indications of heredity.

VI.—NUTRITION AND INTRAOCULAR TENSION.

30. GSTETTNER, MATHILDE. **Experiments with lenses, vitreous, aqueous, and serum with relation to their behavior in the presence of inorganic alkalies and alkaline salt solutions.** *Wiener med. Wochenschrift*, Nos. 30–36.

GSTETTNER (30, **Behavior of lenses, vitreous, aqueous, and serum in the presence of inorganic alkalies and alkaline salt solutions**) placed lenses carefully removed in their capsules from animals in 5*ccm* of physiological salt solution to which had been added 1*ccm* of a 2 to 1000 solution of soda. These withdrew the alkali from the fluid in a short time, so that when phenolphthalein had been added previously the rose red color of the fluid disappeared within two hours. A lens can be used for four successive experiments. The cortical layer of the lens possesses this capacity to a higher degree than the nucleus, and the vitreous to a still higher degree. Cataractous human lenses have the same capacity, and the decolorization seems to be performed more quickly by those from young persons than by those from older ones. The experiment was successful with lenses that had been fixed in alcohol, and the decolorization took only a few minutes when pulverized dry lens matter was used. Alcoholic and etheric extracts of the lens likewise decolorized the fluid. Fresh serum decolorized the fluid even more rapidly than the vitreous. The aqueous, retina, choroid, and ocular muscles acted in the same way. The same was true of the liver and kidneys, and to a less degree of the lungs and heart. The yolks of hens' eggs, whether fresh or boiled, behaved the same as serum, the albumen like the vitreous. A small quantity of grape sugar added to the aqueous did away with the decolorizing power of the lens after about two hours, but was ineffective

in serum even after a longer period. The writer presents these facts without drawing any conclusions.

VII.—THE SENSE OF SIGHT.

31. AUGSTEIN. **Military experiences concerning hemeralopia and the fundus of the eye.** *Klin. Monatsbl. f. Augenheilkunde*, lv., p. 474.

32. BARRIT. **Mental retardation, nutrition, and eyesight in school children.** *Ophthalmoscope*, September, 1915.

33. HEGNER. **Rare forms of hemianopsic disturbances of the field after shot wounds.** *Klin. Monatsbl. f. Augenheilkunde*, lv., p. 642.

34. HEINE. **Treatment of monolateral amblyopia.** *Muenchener medizinische Wochenschrift*, xliii., p. 1453.

35. JAKS. **Treatment of hemeralopia.** *Ibid.*, xlix., p. 1707.

36. PAUL. **Observations concerning hemeralopia in the field.** *Ibid.*, xlv., p. 1548.

37. SCHANZ. **Light filter.** *Ibid.*, xlviii., p. 1640.

SCHANZ (37, **Light filter**) divides the rays of light according to their physiological effects into three groups: those that are directly visible, those that are visible indirectly as fluorescent light, and those that are not visible at all. According to the idea of the writer it is necessary to exclude the second class in order to prevent the formation of cataract, so he recommends the euphos glass again for this purpose. Then he argues against the view that the artificial high sun can take the place of sunlight in treatment with light, and describes the differences in the spectra.

According to BARRIT (32, **Mental retardation, nutrition, and eyesight in school children**) the eyesight of a child is an index to his nutrition. Hypermetropia, which is the normal condition of a child, necessitates accommodation, the tonus or normal state of tension of the ciliary muscle, to obtain good vision. Impaired nutrition, whether through disease or anything else, weakens the tonus of the ciliary muscle, just as it does that of other muscles, and renders the vision poor. In myopia the poor vision is not caused by lack of accommodative power, but it is the index of past or present weakness productive of abnormal conditions which can cause myopia. Two tables are appended; the first shows the nutritive condition of children with different degrees of vision, the second comprises the children who are three years backward in the schools.

HOEVE.

HEINE (34, **Treatment of monolateral amblyopia**) recommends trying the stenopaic hole or cleft, for in many cases the vision can be doubled in this way. Such a correction can play a great part in binocular vision. A horizontally placed cleft often is better than a hole in the interest of horizontal fusion, provided that the horizontal is not the ametropic meridian, when two stenopaic holes three millimeters apart can be substituted. In hypermetropia the vision can be increased considerably by a weak convex glass at some distance in front of the eye, so placed that to a certain degree it resembles the objective of a spyglass. For example, in a hypermetropia of 8 D. the vision was almost tripled by a convex lens of 3 D. at a distance of *21cm.*

AUGSTEIN (31, **Hemeralopia in the army**) has studied fifty-eight cases of hemeralopia that came under his observation during the first year of the war. He found changes in the fundus strikingly frequent, especially in the periphery, by a thorough examination with the ophthalmoscope, which he divides into three groups: (1) Changes which in essentials resemble the grayish white discoloration described by Oguchi; (2) deep black round spots, or black, thick stripes, and depigmentations; (3) a picture like that produced by the atrophy due to stretching in myopia. He thinks it probable that such choroidal changes had been present a long time and did not arise during the war, but that external influences can excite the disturbance of adaptation in eyes that are rendered predisposed in this manner.

PAUL (36, **Hemeralopia in the army**) has often observed hemeralopia in the absence of objective eye symptoms, frequently in persons with labile nervous systems and with psychical depressions, and therefore considers the hemeralopia met with in the army as a cerebral trouble, the result of nervous weakness and psychical depression. The prognosis is uncertain; cure depends mainly on protection from nervous and psychical influences that tend to harm.

For the treatment of hemeralopia in the field JAKS (35, **Treatment of hemeralopia**) recommends iron as more efficient than cod-liver oil.

HEGNER (33, **Rare forms of hemianopsia after shot wounds**) reports three interesting cases of slight injuries of the occipital

bone, each of which caused a hemianopsic scotoma which involved the point of fixation. These homonymous defects in the region of the visual field of the macula were like those described by Wilbrand and, taking into account their origin, form a further evidence in favor of the theory that there is a center for the macula in the cortex of the brain. With regard to the sparing of the macula he points out that the theory of the double supply of the macula is not slightly shaken by these cases.

VIII.—ACCOMMODATION AND REFRACTION.

38. DUANE, A. **Anomalies of accommodation clinically considered.** *Transactions of the American Ophthalmological Society*, 1915.

39. KOEGEL. **Pupillary distance and refraction.** *Zeitschrift f. ophthalm. Optik.*, iii., 5, p. 129.

40. MALLWITZ. **The effect of subconjunctival injections of salt solution in the intraocular complications of high myopia.** *Klin. Monatsbl. f. Augenheilkunde*, lv., p. 365.

41. VOGT. **Regular astigmatism of the cornea caused by an orbital tumor.** *Ibid.*, p. 652.

DUANE (38, **Anomalies of accommodation**) presents a table showing the mean value of accommodation for ages between 8 and 68, also the upper and lower limits. It is based upon 12,000 examinations of 1500 eyes. He classifies varieties of a normal accommodation under five heads: insufficiency of accommodation, difficult or ill-sustained accommodation, inertia of accommodation, inequality in the two eyes, and excessive accommodation.

ALLING.

KOEGEL (39, **Pupillary distance and refraction**) has studied the relations between the refraction and the distance between the pupils in 350 persons. He finds that a greater pupillary distance is demonstrable in myopes than in hypermetropes, while that of emmetropes is intermediate. The average pupillary distance in hypermetropes is 63.81*mm*, in emmetropes 65.1*mm*, in myopes 67.27*mm*. The average figures are smaller in women, in whom they are 62.27, 63.23, and 64.7*mm* respectively. The measurements from the middle of the root of the nose to the center of the pupil were symmetrical in only 13.5% of the men, and 16.4% of the women. No relations could be found between anomalies of the refraction and the

shape of the head. Finally he suggests that it is possible that eyes with a greater pupillary distance have a greater tendency to become myopic because the convergence for near objects must be greater.

MALLWITZ (40, **Subconjunctival injections of salt for the intraocular complications of high myopia**) finds that the results of this treatment are very good as an improvement of vision was obtained in seventy-five per cent. of the cases, number not given. The improvements in vision under the treatment formerly employed were about seventy-eight per cent., but the unpleasant consequences were such that he considers the subconjunctival injection of salt solution to be a marked advance.

VOGT (41, **Regular astigmatism of the cornea caused by an orbital tumor**) reports the case of a man who had had a slowly growing tumor in the orbit since childhood and presented in the corresponding eye a considerable corneal astigmatism, which was not present in the other. The form and degree of the astigmatism led him to conclude that it had been caused by the pressure of the tumor.

IX.—THE MOTOR APPARATUS OF THE EYE.

42. COULTER, R. J. **Nystagmus cases and electric lights.** *Ophthalmoscope*, November, 1915, p. 556.

43. GREY, E. G. **Studies on the localization of cerebral tumors.** *Journal A. M. A.*, October 16, 1915.

44. HOENIG. **Oculomotor paralysis as the first symptom of an abscess of the frontal portion of the brain.** *Klin. Monatsbl. f. Augenheilk.*, lv., p. 382.

45. KOERNER. **The position of the eyebrows in peripheral facial paralysis.** *Zeitschrift f. Ohrenheilk u. f. d. Krankh. d. Luftwege*, lxxii., 4.

46. MARBURG. **Apoplectiform bulbar paralysis.** *K. k. Gesellschaft der Aerzte Wien*, November 22, 1915.

47. POSEY, W. C. **Tenotomy of the inferior oblique muscle.** *Transactions of the American Ophthalmological Society*, 1915.

POSEY (47, **Tenotomy of the inferior oblique muscle**) reports seventeen cases in which this operation was performed. The most common type consisted of an insufficiency of the superior rectus in one eye with overaction of the inferior oblique of its fellow. The next most frequent form was insufficiency of both superior recti with overaction of both

inferior obliques. Other cases were of mixed type. The technic consists in making an incision through the skin down to the bone just above and parallel to the lower edge of the orbit. A strabismus hook is then introduced and the muscle caught and cut at its origin in the floor of the orbit. The results were all satisfactory. The up and in tilt imparted to the eye by the overaction of the muscles generally disappeared. In all but one case the defect was congenital.

ALLING.

GREY (43, **Localization of cerebral tumors**) states that all authorities agree that nystagmus is a valuable localizing sign of disease in the posterior fossa. In fifty-one cases of verified tumor in this locality eleven showed no nystagmus. In all cases in which it was absent the lesions were intracerebellar.

ALLING.

HOENIG (44, **Oculomotor paralysis the first symptom of a frontal abscess of the brain**) saw a left-sided oculomotor paralysis as the first and only focal symptom in a case of this nature. At first the paralysis was limited to all of the outer twigs of the oculomotorius, but after some weeks the inner twigs were involved. Four days before death a slight nystagmus was added. The writer was unable to make up his mind whether the paralysis was the consequence of pressure upon either the left lateral ventricle, or the caudex cerebri, or the result of a toxic affection of the nucleus.

In consequence of a shot wound from the forehead to the occiput a man described by MARBURG (46, **Apoplectiform bulbar paralysis**) suffered from oculomotor paralysis with amaurosis on one side with a paresis of the facial, the trigeminus, and the trochlearis on the other. The vagus and hypoglossus also were affected, so the picture presented was that of an apoplectiform bulbar paralysis.

In two cases of otogenous facial paralysis KOERNER (45, **Position of the eyebrows in peripheral facial paralysis**) saw that the eyebrow on the affected side was on a lower level than on the other, the difference in the height of the two being about twelve millimeters when the head was held erect. He explains the phenomenon by saying that many people are accustomed to keep their occipito-frontalis strongly contracted.

COULTER (42, **Nystagmus cases and electric lights**) in dis-

cussing nystagmus in this communication draws the following conclusions:

1. That insufficient light causes nystagmus.
2. That the introduction of electric lights has enabled scores of our workmen to continue working underground.
3. That the use of electric lamps is a good preventative of nystagmus.

CURRAN.

X.—LIDS.

48. BEDELL, A. J. **Report of a case of colloid degeneration of the upper lid.** *Transactions of the American Ophthalmological Society*, 1915.

BEDELL'S (48, **Colloid degeneration of the upper lid**) patient showed the whole upper lid rounded out by a firm mass of cartilaginous consistence involving the conjunctiva and tarsus but not the skin. About one-half of the conjunctiva of the lid was unaffected and after enucleation of the growth was brought together by sutures. Under the microscope the specimen appeared to consist almost entirely of structureless masses—amyloid degeneration.

ALLING.

XI.—LACRIMAL ORGANS.

49. ERB. **Contribution to Toti's dacryocystorhinostomy.** *Festschrift on the Fiftieth Anniversary of the University Eye Clinic of Basel.*

50. KUGELBERG. **Some points in extirpation of the sac.** *Klin. Monatsbl. f. Augenheilkunde*, September–October, 1915, p. 402.

51. RAUPP, R. **Suture of the canaliculus.** *Ibid.*, p. 388.

RAUPP (51, **Suture of the canaliculus**) recommends the following method of repairing a wounded canaliculus as simple and efficient. First sutures are placed in the conjunctival and skin margins of the wound; then one needle of a double armed fine, black silk thread is passed through the proximal opening of the wound in the canaliculus for about three millimeters and then out in the intermarginal space; the other needle is then introduced into the distal portion of the wound and carried along the canaliculus to the punctum where it is brought out. The thread is made tense and the surfaces of the wound are brought into apposition so that when the thread is

tied the parts lie in a normal position without pressure. Several lid sutures may be necessary. Ointment is then applied. Healing is said to take place with perfect cosmetic and functional results.

KUGELBERG (50, **Extirpation of the sac**) makes the points that when local anæsthesia is employed the œdema produced by the injection should be massaged away in order that the normal anatomical landmarks may be brought again into view, as well as that the fluid may be distributed better and deeper, and hemorrhage prevented more perfectly. The incision in the skin and soft parts should extend very little above the internal palpebral ligament in order to avoid the formation of a cicatricial cord in the skin about the canthus.

ERB (49, **Toti's dacryocystorhinostomy**) reports fifteen cases and concludes that this operation, when performed in exact accordance with the directions of its author, gives an ideal functional result, provided that the cases are selected rightly. Only such cases are fitted for the operation as have received no former treatment, or at any rate it is contraindicated in cases that have been treated by probing, or in which one or both canaliculi have been slit. Total extirpation of the sac, perhaps with extirpation of the palpebral lacrimal gland, is indicated in such cases. Total extirpation is the operation of choice also when an operation on the eyeball is to be performed and there is a purulent dacryocystitis. He says that dacryocystorhinostomy is technically no more difficult, but is more irritating than extirpation of the sac, and takes considerably more time.

XII.—ORBITS, EXOPHTHALMOS, ACCESSORY SINUSES.

52. COLE, H. F. **Total removal of the orbit under local anæsthesia.** *Journal A. M. A.*, Dec. 4, 1915.

53. DE SCHWEINITZ, G. E. **A contribution to the subject of tumors of the eyelid and orbit.** *Trans. of the American Ophthalmological Society*, 1915.

54. STARK, H. H. **Sudden blindness due to suppuration of the accessory nasal sinuses.** *Journal A. M. A.*, October 30, 1915.

COLE'S (52, **Total removal of the orbit under local anæsthesia**) patient was eighty-one years old and a subject of nephritis. She had a sarcoma of the conjunctiva of the lower lid. He exenterated the orbit without pain or difficulty by repeated

injections of novocaine-adrenalin solution. He also thoroughly cleaned out the supraorbital and infraorbital canals.

ALLING.

DE SCHWEINITZ (53, **Tumors of the eyelid and orbit**) presents four cases.

Case I. Myxosarcoma of the right eyelid of unusual size, weighing 267 grams. No operation.

Case II. Small round-celled sarcoma of the orbit. Evisceration was done and no recurrence after seven months.

Case III. Final history of intradural tumor. Psammoendothelioma of the optic nerve (previously reported). Eyeball and tumor removed. Recurrence in six months. Complete evisceration. Now some evidences of recurrence in the cranial cavity.

Case IV. Epibulbar lipoma. Chief interest in the size, the tumor extending from near the limbus over the upper and outer portion of the eyeball.

ALLING.

STARK (54, **Sudden blindness due to suppuration of the accessory nasal sinuses**) has observed three cases of this sort cured by operation. One had central scotoma for red and green, one dilated pupil, and one dilated pupil, color scotoma, and involvement of the external rectus. He has collected from literature eighty-eight cases of rapid onset. In nineteen both eyes were affected. The diagnostic signs are discussed. They include optic neuritis, dilated or contracted pupils, exophthalmos, contraction of the field, central scotoma, and enlargement of the blind spot.

ALLING.

XIII.—CONJUNCTIVA.

55. CAPRON, F. P. **Multiple melanotic nævi of the conjunctiva with malignant invasion of the corneal limbus.** *Trans. Am. Ophth. Soc.*, 1915.

56. DERBY, G. S. **Some aspects of ophthalmia neonatorum.** *Journal A. M. A.*, October 16, 1915.

57. DERNEHL, P. H. **Carcinoma of the caruncle with report of a case.** *Ophthalmology*, October, 1915.

58. EYMANN, L. **Contact carcinoma of the palpebral conjunctiva and of cornea.** *Klinische Monatsblaetter f. Augenheilkunde*, September-October, p. 339.

59. MUSY. **A case of Parinaud's conjunctivitis of tuberculous origin.** *Festschrift of the Fiftieth Anniversary of the University Eye Clinic at Basel.*

60. STIEREN, EDW. **Pemphigus of the conjunctiva. Report of two cases.** *Transactions of the American Ophthalmological Society*, 1915.

MUSY (59, **Parinaud's conjunctivitis of tuberculous origin**) reports a case that he took to be one of Parinaud's conjunctivitis, but demonstrated the disease to be tuberculous through inoculation of the anterior chamber of a rabbit and of the peritoneum of a guinea-pig. He was unable to determine whether the tuberculosis was of the human or the bovine type.

EYMANN (58, **Contact carcinoma of the palpebral conjunctiva and of the cornea**) describes a patient eighty-two years old who had a small, rough, ulcerated growth on the conjunctiva of the upper lid which pressed upon the eyeball. The lateral two-thirds of the cornea, together with the adjacent bulbar conjunctiva, was covered with reddish granulation tissue. Toward the fornix the conjunctiva showed nothing that resembled an infiltration. Macroscopically there was no unbroken connection between the thickening of the upper lid and the corresponding thickening on the cornea and adjacent conjunctiva. The tumor of the upper lid proved under the microscope to be a typical epithelioma of the flat cell or basal cell type. The surface of the eyeball which was in contact with the tumor of the lid showed a distinct carcinomatous proliferation of the epithelium of the cornea and of the adjacent conjunctiva, while any direct connection between the two was thoroughly excluded. Toward the fornix the tumor was distinctly delimited by connective tissue.

DERNEHL (57, **Carcinoma of the caruncle**) has found only five authentic cases of this sort on record. His case was a man of fifty-six who presented a brownish tumor mass practically replacing the caruncle of the right eye. It measured about 6 by 7*mm.* The caruncle of the left eye was slightly enlarged, firm, and of bright red color. Both tumors were removed and proved to be basal-celled epitheliomata or hair-sheath carcinomata. This type is often multiple but does not metastasize.

ALLING.

CAPRON'S (55, **Multiple melanotic nævi of the conjunctiva**) patient had had for many years pigment deposits in the palpebral conjunctiva. One spot began to enlarge and proved to be sarcoma. Later another growth appeared at the limbus and

the eye was enucleated together with all but two of the melanotic areas.

ALLING.

One of the cases reported by STIEREN (60, **Pemphigus of the conjunctiva**) was a syphilitic, who suffered from dermatitis bullosa of the entire body. There was entropion of both lower lids. The conjunctiva was glistening and shrunken, having a number of adhesions to the eyeball. Both corneæ were cloudy —the left showing what appeared to be an internal pterygium. A canthoplasty was done in each eye and the symblepharons were turned in, the result being quite satisfactory. The second case was a child of six who developed a bullous eruption over the body four weeks after vaccination. The conjunctiva was shrunken and there were numerous bands of adhesions which were dissected up and the defects covered by sliding flaps. The corneæ were opaque and later became degenerated so that the child was blind. Fresh bullæ appeared on the body from time to time with rises in temperature. A diplococcus larger than the gonococcus was obtained from the serum of the bullæ. Noguchi and Wassermann tests were negative.

ALLING.

DERBY (56, **Ophthalmia neonatorum**) bases his paper on the study of 149 cases. In spite of the dangers attendant upon feeding and the lowered vitality of these infants the mortality of his cases was only 8%. Seventy-seven of all cases were definitely due to the gonococcus, eighteen were monocular, and in no one was the other eye affected after admission to the hospital. He calls attention to the fact that the examination of smears is not conclusive when a Gram negative intracellular diplococcus is found. Cultures should be taken and if a growth results, the organism is not the gonococcus but may be the micrococcus catarrhalis. He thinks that the reason why the gonorrheal conjunctivitis in infants is less severe than in the adult is because there is a certain degree of immunity transmitted from the mother to her offspring. In most cases, however, this is not sufficient to produce a complement-fixation reaction. He pleads for social service work among the parents to prevent dangers inherent in their condition and to future children.

ALLING.

XIV.—CORNEA AND SCLERA.

61. BROWN, S. H. **Trophic ulcer of the cornea due to fracture of the jaw.** *American Journal of Ophthalmology*, October, 1915.

62. DERBY, G. S. **Massive granuloma of the sclera (brawny scleritis) with the report of an unusual case.** *Trans. Amer. Ophthalm. Soc.*, 1915.

63. HANSELL, H. F. **A case of epibulbar sarcoma.** *Ibid.*, 1915.

64. LANE, F. **Peripheral annular infiltration of the cornea following a scleral perforation.** *Annals of Ophthalmology*, October, 1915.

65. STEINORT, A. **Epithelial growths of the cornea.** *Klinische Monatsblaetter f. Augenheilkunde*, September-October, 1915, p. 325.

66. UHTHOFF, W. **Further clinical and anatomical contributions to the degenerative changes of the cornea.** *Ibid.*, p. 290.

67. VERHOEFF, F. H. **Rosacea keratitis and certain other forms of marginal keratitis neuropathic in origin. Treatment by pericorneal neurotomy.** *Transactions of the American Ophthalmological Society*, 1915.

UHTHOFF'S (66, **Degenerative changes of the cornea**) first case was one of Groenouw's nodular degeneration of the cornea in a man thirty-seven years old. The cornea presented, especially in the center, numerous little, grayish, subepithelial foci of various sizes, over which the epithelium everywhere was intact and lustrous. A superficial piece of the lamellæ of the cornea was removed for microscopic examination and showed the degenerative changes in a very early stage, before the deposit of hyaline masses. The pathological condition appears in the region of Bowman's membrane, which was split up in the form of proliferations both in front of and behind this membrane with involvement of the corneal corpuscles, which were enlarged and swollen and showed in places a distinctly finely granular protoplasm. At some places Bowman's membrane was destroyed and the proliferating tissue pressed forward, spreading between this membrane and the epithelium. There was no peculiar proliferation of the epithelium, but on the contrary a thinning through atrophy of the cylindrical basal cells. The case was cured by the ablation of the superficial layers of the cornea. The second case Uhthoff designates as a rare one of gray spotted degeneration of both corneæ. The patient was thirty-four years old. The little gray, conical formations extended from the subepithelial parts of the cornea down deep into the most posterior layers. The chronic degenerative character of the changes was indicated by its appearance in both eyes, the absence of

marked inflammatory symptoms, and the absence of change during a long period of observation. The picture differs from that of nodular keratitis in the form of the little conical formations, which are not regularly arranged, and not chiefly in the center and the region of the palpebral fissure, and also in the absence of the fine, grayish stippling of the parts of the cornea that lie between them. The foci run sagittally deep into the stroma in the form of conical fibrillary degeneration of the corneal tissue with proliferation of the corneal corpuscles. In the middle of one of these a slender canal-like hollow space occurred. Bowman's membrane is affected very little or not at all, and no hyaline deposits could be found.

In STEINORT'S (65, **Epithelial growths of the cornea**) case the tumor occupied nearly the entire cornea and overlapped the sclera above. Its structure was that of an epithelioma, though an active proliferation of connective tissue out from the cornea was not to be mistaken. As the original seat of the tumor was distinctly corneal, and the carcinomatous structure nowhere passed the limbus, it can be spoken of correctly as a primary epithelioma of the cornea. The neighboring parts of conjunctiva and cornea showed a papillary structure. The epithelioma itself had a certain resemblance to the so-called basal cell cancer, including the active involvement of the connective tissue. Such primary epitheliomata do not start from the limbus, but from the cornea itself.

VERHOEFF (67, **Neuropathic marginal keratitis**) is convinced that the lesions seen on the cornea about 1½*mm* from the periphery and associated with herpes facialis are of neuropathic origin. They occur at the termination of the conjunctival nerves of the cornea. He has operated upon fifteen cases by a pericorneal neurotomy, thus cutting off the impulses which he assumes come from the Gasserian ganglion, and has had uniform success.

ALLING.

BROWN'S (61, **Trophic ulcer of the cornea due to fracture of the jaw**) patient had a fracture of the upper and lower jaw on the left side as well as the base of the skull and in a few days developed infiltration and ulceration of almost the entire left cornea. There was anæsthesia of the whole eyeball and impaired sensation over the distribution of the terminal

branches of the fifth nerve. The condition was apparently due to incarceration of the fifth nerve in the callus thrown out in the course of the union of the fracture of the base of the skull.

ALLING.

LANE (64, **Peripheral annular infiltrate of the cornea following a scleral perforation**) examined an eye which had been removed on account of a septic inflammation following a perforating wound. Besides the usual signs, it showed a millimeter-broad, gray ring of deep infiltration encircling the cornea with its peripheral edge 1 to *2mm* from the limbus. Under the microscope the cornea in this zone showed the lamellæ spread apart by wide lacunæ closely packed with polynuclear leucocytes. The cornea was thus thickened and the surface projecting with Bowman's membrane thinned. The infiltration stopped abruptly on the peripheral side but disappeared gradually toward the center of the cornea. Lane has examined 465 eyes enucleated after perforation and this is the first one in which the condition described has been found.

ALLING.

The tumor described by HANSELL (63, **Epibulbar sarcoma**) was the size of a marble and attached to the sclera about *8mm* from the limbus. In excising the growth, which was adherent only at the center of its base, the sclera was punctured. It was a round-celled sarcoma.

ALLING.

DERBY'S (62, **Brawny scleritis**) case was characterized by a chronic inflammatory process which had lasted for five years. The appearances were those of a mild scleral inflammation with accompanying uveitis and engorgement of the scleral veins. The eye was soft but there was no sign of shrinking. The pathologic process was an immense thickening of the sclera caused by new formation of dense fibrous tissue. Within the tissue were areas of purulent infiltration, caseation, and marked obliterative endarteritis.

ALLING.

(To be continued.)